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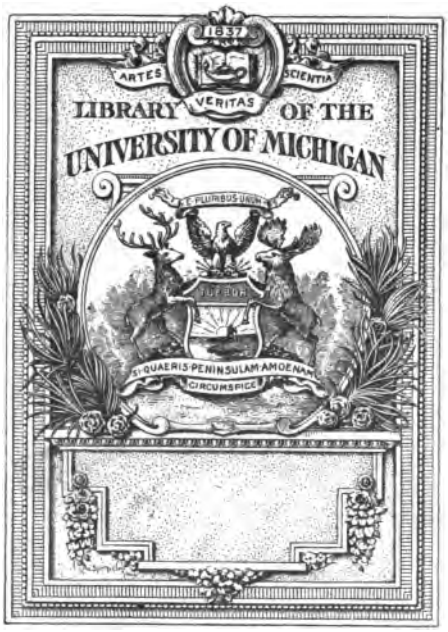
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WESTMINSTER HOSPITAL REPORTS.

EDITED BY

OCTAVIUS STURGES, M.D.Cantab., F.R.C.P.,

AND

GEORGE COWELL, F.R.C.S.

VOLUME VI.

LONDON:

J. & A. CHURCHILL,

11, NEW BURLINGTON STREET,

1890.

Published at 6s. Price to Subscribers, 5s.

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ORIGINAL ARTICLES.

In Memoriam.

DR. GEORGE TUPMAN FINCHAM.

By GEORGE COWELL.

Our volume of Reports unfortunately again opens with a record of a much-regretted loss sustained by our staff—a loss which is tempered, however, by many cheering and enduring memories. The latest of our Consulting Physicians has gone to his rest, and it is our duty, whilst expressing the sorrow universally felt by colleagues and pupils alike, to endeavour, however imperfectly, to express in words some account of that life which is gone from us, and of that life's work and character which will live in the hearts of all who knew him.

Dr. Fincham, like Dr. Radcliffe, died somewhat suddenly. It had been noticed, however, that he had been failing in health for some months, and, as he suffered from aortic insufficiency, his friends were not free from anxiety about him. He had retired from practice some twelve months before, but he used still to show himself at the College of Physicians, and often attended the meetings of the House Committee of the Westminster Hospital, in the affairs of both of which institutions he continued to take the warmest interest. The immediate cause of death was probably the giving way of a small cerebral vessel, for he was found insensible and died a few hours afterwards, on May 26th, 1890, in his 73rd year.

George Tupman Fincham was born on January 28th, 1818, at a house in Spring Gardens. He was the son of a medical man,

Mr. George Fincham, in general practice at that place. He was sent to Westminster School in 1827, and later, in 1831, was transferred to King's College School, then under the Head Mastership of the Rev. Dr. Major, who was a connection by marriage of his mother. In 1834 we find him in the Higher Department of the School at work at Latin and Greek. In 1836 he matriculated at St. John's College at Oxford, whither he went up to reside in October of that year. Here he entered with zest into that study of scholarship and Greek and Latin literature, happily held to be essential to the cultured Physician of those days. In due course he graduated in Arts, and, having chosen his father's profession, he returned to London and entered at St. George's Hospital in October, 1839, where he studied Chemistry under Mr. Faraday and Mr. Brand. A year later he went to Paris, and studied medicine during the winter, under Professor Chomel, and established a friendship with Dr. Noel Gueneau de Mussy, who was at that time Dr. Chomel's Chef de Clinique. He then returned to St. George's, where he worked steadily and very successfully until the summer of 1842, when he went to Dublin and became a resident pupil at the Rotunda. In that city he also attended the practice of that scholarly Physician, Dr. Graves. After another winter at St. George's he passed the examination for the M.B. Degree at Oxford in 1843, and in the following year became a Member—or, as it was then called, Licentiate—of the Royal College of Physicians.

Dr. Fincham's first spheres of public practice were the Public Dispensary in Bishop's Court, afterwards in Carey Street, Lincoln's Inn Fields, and the Western Dispensary, at both of which institutions his work speedily showed the painstaking and conscientious character of the man. The M.D. degree at Oxford was taken in 1847, in which year also he married Miss Heygate, the daughter of James Heygate, Esq., of Southend, Essex.

His first connection with Westminster Hospital was as Lecturer on Forensic Medicine in the Medical School, to which Chair he was appointed in 1850, and which he held until 1865. On January 22nd, 1853, he was elected, at the age of thirty-five, coadjutor to Dr. Woodfall, the first Assistant Physician appointed to the Hospital, by whose resignation, however, in May of the same year, followed by Dr. Kingston's retirement two years later, he rapidly became in succession Senior Assistant Physician and

full Physician, to which latter post he was appointed on April 21st, 1855. Dr. Fincham was elected a Fellow of the College of Physicians in the same year. Here also he was held in high honour, and was appointed Censor in 1872-4 and in 1877-8, Councillor in 1876, 1883, and 1885, Examiner in 1881-2, Senior Censor in 1883-4, and in 1884 the senior of the two first Vice-Presidents that were appointed.

Dr. Fincham had been twenty years full Physician at the Hospital before he became in any way connected with the Chair of Systematic Medicine. This Chair was held for many years by the late Dr. Basham, and when he resigned it in 1871 Dr. Fincham, with his usual unselfish conscientiousness, persuaded himself that it was to the advantage of the Medical School that Dr. Anstie should succeed to it, and himself declined the post. However, on the death of Anstie in 1875, it was thought important to the School that he should step into the breach, and he most kindly undertook to share the Chair with Dr. Sturges, and continued to deliver half the Lectures on Medicine for eight years, until 1883, when by an inexorable rule he had to retire from the post of Physician of the Hospital and was elected Consulting Physician, and Westminster regretfully lost his valuable services. He however retained his seat on the House Committee, the deliberations of which he continued to attend until within a few days of his death. He twice delivered the Introductory Lecture at the opening of the Winter Session—in 1856 and 1866. From 1853 to 1889 he was Physician to the Scottish Provincial Life Assurance Company, and for some years he was Physician to the Westminster School. He leaves a widow, two sons, and two daughters.

Dr. Fincham was one of that good old type of scholarly Physicians, the disappearance of which from amongst us is a great misfortune to our profession. Trained himself at Oxford by the study of scholarship, and refined by acquaintance with the best literature of Rome and Greece, a taste which he always maintained, there was hardly any subject that pained him more than the neglect of the classics, as the foundation of mental training and development. He always held that the training of the mind was something apart from mere teaching, and was strongly convinced that the study of Latin and Greek afforded the best means to this end. Science was ill-adapted, even if of any real

service, to be a means of training, whilst it was certain that a knowledge of it could be far more easily acquired after the mind had been properly developed by scholarship or mathematics. How completely opposed to Dr. Fincham's opinion have been the recent ill-considered pronouncements in favour of abolishing altogether Latin and Greek as a preliminary training for medical men.

On a foundation, then, of sound learning and much literary culture, Dr. Fincham had built a good practical knowledge of medicine. In applying this knowledge at the bedside, he was ever most cautious, conscientious, and painstaking, sparing neither time nor patience in his endeavour in obscure cases to arrive at a correct diagnosis. Any striving after effect was entirely absent from his thoughts, and in fact opposed to his nature; there was only an honest endeavour to obtain a correct view of the case before him, stimulated by an eager interest innate in the true physician. But it must not be thought from what has been said that Dr. Fincham was slow in forming his opinion of a case. With his habitual care and caution, he possessed, perhaps without knowing it, for he never himself relied upon it, a true sagacity and clinical penetration which could not fail to impress his pupils and those whom he met in consultation. Still he endeavoured to confirm and verify the suggestions of his sagacity, and did not assert that of which he was not himself convinced. Dr. Fincham combined with this conscientious care a remarkable tenderness for and sympathy with his patients, which often gained the affection as well as the confidence of hospital and private patients alike. That this tenderness and sympathy were born of a recognition that he had not merely to treat a disease but a sick person, was shown in the thoughtful kindness with which his opinion was conveyed, in the judicious selection of the treatment prescribed, and in the delicacy of feeling, and yet firmness, which always characterised the advice and warnings that he had to give. Doctors are not agreed as to the duty of warning a patient of his approaching end, but Dr. Fincham held most strongly that this duty was paramount.

Dr. Fincham was too cautious and not sufficiently dogmatic to be considered a brilliant teacher, but yet in reality he was one of the best of teachers. It is said that the art of taking pains is the

secret of success. How many of Dr. Fincham's pupils must thank him for the acquisition of this art. It will be an evil day for medical training when all teachers are dogmatic, or when all teachers follow the same methods. Dogmatic teaching is of great service to the student who is preparing for examination, but it is in itself not sufficient to make him a successful practitioner. Students must learn to think and reason for themselves, and Dr. Fincham's method was well calculated to this end. And the lessons that were learnt from him, how to observe disease, how to interrogate patients, how to interpret and appreciate symptoms —in short, the methods by which an accurate diagnosis may be obtained—will not easily be forgotten by those who had the good fortune to be his pupils. His kindness of manner, and the devotion and conscientiousness with which he ever performed his hospital duties, will live, too, in the recollection of all.

Dr. Fincham was not often seen at the medical societies, and his published writings were very few. Of the latter, I believe that the following is a complete list:—

'A Lecture on Clinical Teaching and Clinical Study,' *Medical Times and Gazette*, 1862.

'A Clinical Lecture on a case of Aortic Aneurism,' *Medical Times and Gazette*, 1864.

'Observations on Diarrhœa and Cholera,' *British Medical Journal*, 1871.

'Pallor of the Skin in some of its Clinical Aspects' (the substance of two Clinical Lectures), *Westminster Hospital Reports*, vol. i., 1885.

Thus far I have confined myself to the professional history and character of Dr. Fincham, and one hesitates to go beyond it. And yet how miserably imperfect would be this notice if it failed to give one glimpse of his private life and character, and of the tastes and feelings that influenced and harmonised his professional and domestic life. He was a man of strong affections, and thoroughly understood the full meaning of the word *Home*. It therefore goes without saying that he was, in the best sense, a good husband and father. He made warm friendships, too, with such of those with whom he was brought much in contact, who succeeded in gaining his respect; and his high moral tone, his nobility of character, his unswerving integrity, his unassuming modesty of bearing, and his charm of manner, never

failed to attract and influence those who were capable of appreciating them.

The principle that may perhaps be said to have influenced his whole life was his respect and veneration for authority. It was this that made him ever loyal to his profession, his college, and his hospital. It was this that explained his reticence. His devotion to the discharge of duty excluded all selfishness and self-seeking, and he wrote not to push himself forward, but only when he had something that ought to be said. It was this that made him a Conservative in politics, and rendered it impossible for him to have any sympathy with the modern methods of political controversy, and the vain theories propounded for so-called socialist reforms. It was this, too, that led him in 1855 to seek admission into the Roman Catholic Church, as he failed to satisfy himself that the English Church possessed the necessary authority.

Dr. Fincham did not go much into society, and most of his extensive reading was done aloud at his own fireside and to the members of his own family. The many pleasant conversations on these books prevented the subjects of them from being forgotten, and many happy memories of these are still cherished by those who participated in them.

It has been wisely said that no man can thoroughly enjoy and appreciate a holiday, who, first, is not accustomed to throw himself heart and soul into his work, and, secondly, does not possess some favourite study or adopt some attractive pursuit outside his ordinary work. Such means of cultivation and relaxation Dr. Fincham found in the fascinating study of Architecture, and especially in its ecclesiastical examples. Amongst his books were many on this subject, and for years it determined the direction of his holidays both at home and on the Continent. He never tired of wandering about those cities and towns, whether in England or abroad, which possessed noble churches, those poems in stone which demonstrated the faith and aspirations of the builders, and whose beauty and proportions spoke to him with no uncertain voice. And when he returned to home and work he still would delight in describing what he had seen, and the minsters that had entranced him, warming the listener to the desire of visiting the same marvellous structures and elevating him to a like admiration of them with himself.

It was a happy thought, when, on Dr. Fincham's retirement from the post of Physician at the Hospital, and when he was feeling the wrench of being deposed from the happiest work of his life, the spontaneous desire to give expression to their feeling of regret and loss, led the subscribers to include in the presentation some etchings of exquisite architectural examples. It was this perhaps more than anything else that showed the genuineness and sincerity of the feelings so unanimously expressed, and which overwhelmed him with emotion. It is rarely that a man retires from his life's work amid such widespread regret or so universally beloved.

REQUIESCAT IN PACE.

OF PROGNOSIS IN PNEUMONIA.

By Dr. STURGES.

It is often said that the knowledge of disease gained at the bedside can be got in no other way, and confers something akin to prophetic insight. If this be true speaking generally, and especially true, as is also affirmed, of the specific fevers, it is certainly to be questioned as regards a disease which if not itself a fever is at least fever-like, namely, pneumonia. That long experience of pneumonia quickens the faculty of prognosis I very much doubt. There are few affections whereof the forecast is so often erroneous, and what practice gives is not so much insight as caution.

The last two years—the period, that is, which covers the influenza epidemic—have been rich in examples of what we call anomalous pneumonia, meaning thereby cases that err in some respect or other from our preconceptions, and are thus described to excuse our false estimate of them and save our credit. Many examples in point might be given from our Hospital Records. They suggest above all the reflection that the knowledge of pneumonia, though undoubtedly growing, is still very limited; that it is not to be reckoned a disease, but rather a symptom common to many diseases. By way of illustration I proceed to give very shortly the leading features of two cases lately under my charge, the patients both lads of nineteen, of previously good health, and both exhibiting the physical and other signs of acute lobar pneumonia.

Case 1.—Henry B., aged 19. Hotel plate cleaner, pale, and of rather slender build; was admitted into Hallett Ward, June 27th, 1890. He was in his usual good health until a fortnight before admission, when, having taken cold as he supposed, he shivered, but had no pleural pain. Next day cough came on; he felt hot and sweated. He still kept at his work, though

distressed from dyspnœa. A week before admission (that is, a week after the rigor) he came to hospital as an out-patient; but no physical signs of lung inflammation were then discovered. On the 27th he came again, and was admitted. Impaired resonance with medium crepitus was then found over the left base; temperature was 100°; skin hot and moist; cheeks slightly flushed; tongue coated; respirations 34; pulse 128.

His personal and family history were good, except that his father was said to have died of consumption. He had himself had measles and whooping cough as a child, but not rheumatic or scarlet fever or any lung affection.

On the day after admission the morbid signs applied to both bases, dulness, namely, with numerous medium crepitations, and at the right base, bronchial to tubular breathing with some pleural friction; temperature 102·8°; pulse 128; urine sp. gr. 1027. No albumen. Some dark-coloured, not characteristic sputum.

During the next week the patient remained much the same, the right base respiration becoming distinctly tubular; respirations varying from 36 to 40, and pulse between 116 and 130. Temperature during this period—June 24th to July 4th—ranged from 100° to 103°, the evening being usually the higher.

As time went on, the only change in the physical signs was marked ægophony over the right base, where the breathing was still tubular and medium crepitus was still audible. The left base still remaining dull, yielded bronchial rather than tubular breathing, and the voice was less distinctly ægophonic than at the right base. (A syringe exploration was made at a carefully selected spot in the right axillary line, but no sign of fluid was obtained.)

At the end of three weeks the general condition was not much altered, except that the boy was weaker. The main change in the physical signs was in the sharper and more ringing character of the crepitus, breathing being still tubular over portions of both lungs, a condition strongly suggestive of commencing softening.

On August 16th, after seven weeks residence, signs of consolidation were audible at both apices, and the left base, which in the interval between July 15th and August 15th had almost cleared up, again became dull and yielded bronchial, almost tubular breathing. Pyrexia was still present, and the boy had

lost nearly three pounds in weight. Thus both physical and general signs nearly resembled phthisis.

At this point it will be convenient to pause for a moment, and, before completing the narrative, turn to the other patient.

CASE 2.—Albert S., aged 19. A cellarman, a stout, florid youth, admitted July 14th. His health is generally good, excepting that for three years he has had cough on and off, chiefly in winter, and that nine months ago he was out-patient with symptoms which he describes as resembling the present. There is no family history of phthisis, but his brother is weakly; he has himself had no serious illness. He, too, shivered a week before admission, and got dry, hacking cough with dyspnœa. He had also, as the other had not, pain in the side, and two days before admission vomited and became acutely ill. He attributed his attack to getting wet and remaining in his wet clothes for the whole day.

On admission he appeared drowsy and complained of stitch in right side. Tongue was coated, and temperature 101·4°; respiration 38; pulse only 90; urine sp. gr. 1023. No albumen. There was no morbid sound audible in the chest at this time save slight sibilus. Vocal fremitus and resonance were equal for the two sides, but *at the right base breath sounds were very indistinctly heard.*

For the first four days no more distinctive physical signs could be made out, but he continued very drowsy, and the aspect, respiration, and distinctly pneumonic sputum left no doubt as to the nature of the illness. The temperature curve of these four days was peculiar, resembling that of septicæmia, ranging, that is to say, daily from subnormal to 103°, 103·5° or 104°. On the fourth day from admission the pulse (heretofore 90 and under) rose to over 120. By the fourth day pleuritic friction was audible, and soon distinctly tubular respiration and all the usual physical signs of lobar pneumonia. On the fifth day of admission in the evening the temperature settled down to 102°, and there remained until the following evening, when crisis occurred. The critical termination of this pneumonia was thus seven days after the severe symptoms, but twelve days after the initial shivering and dyspnœa.

The above are but short abstracts of the Medical Registrar's

notes, yet they include all that is pertinent to the question I would discuss, namely, the material available for prognosis in pneumonia. The patients may be thus compared.

As regards mode of origin. The first patient (A) had been ill a fortnight with commencing rigor but without "stitch"; a week before admission no physical signs were discoverable. He *thought* he must have caught cold, but knew not how. He had been previously healthy, but his father, it was believed, had died of phthisis.

The second patient (B) had been ill eight days, but an exacerbation marked by vomiting and stitch occurred two days before admission. There was a very definite history of chill due to sitting all day in wet clothes. Though of healthy family, and himself well, he had been subject for some years to winter cough, and nine months before had an attack resembling the present.

As regards pyrexia. In A, the moderate, and, for pneumonia, exceptionally low temperature of the commencement persisted, and such variations as occurred did not correspond with any change in his general condition.

In B the temperature tracing was peculiar in its wide daily range (98° to 104°). It resembled in fact that of septicæmia during the first five days, when it descended critically with the rapid recovery of the patient.

As regards pulse. In A it ranged between 120 and 130, and so continued. In B for the first two days the pulse was under 90; for the next three it was at and about 120, when crisis happened.

From the temperature, the pulse rate, the time of crisis, and the development of physical signs, it was highly probable that this boy was admitted on the second day of pneumonia; and although the history told of eight days' acute illness, it also showed a marked exacerbation two days before admission, which would harmonise with this view. Anyhow, on the seventh day according to one reckoning, on the thirteenth according to the other, the disease reached its term.

As regards physical signs. In A the pneumonic inflammation was double, and for some days it continued to spread. Later on as will be presently seen, it left the left side, but persisted in the right.

In B the physical signs were one-sided, appeared late, and at first amounted to no more than a silence at the affected base.

Thus, if we place side by side the symptoms common to both patients, and the symptoms proper to each, it appears that, as regards antecedents, there was little to choose between them. A was reported to have had a phthisical father; B was himself subject to winter cough, and apparently had had pneumonia before. As regards actual attack, both were suddenly seized with acute symptoms suggesting pneumonia, and both shortly revealed physical conditions indicative of that affection.

The most obvious differences between them were these.

1. A had no definite history of chill.
   B had a most distinct history of it.
2. A had no pleural stitch.
   B had marked pleural stitch.
3. A had never characteristic rusty sputum.
   B had such sputum.
4. A had a moderate sustained temperature.
   B had high and intermittent temperature.
5. A had double pneumonia.
   B had single pneumonia.

Now if these two cases be compared with the set description of pneumonia, with pneumonia as it ought to be, neither of them comes up to the pattern provided, nor is it easy to say whether of the two departs from it most, or to form any prognosis favourable to the one rather than the other. Even in such features as are common to both, neither patient altogether obeys the rule of the book. Observe especially the difficulty in respect of date of invasion. In A it occurred a fortnight before admission, the earliest physical signs being some days later. It far outlasted therefore the ordinary term of pneumonia, and at no time exhibited anything resembling crisis. In B either crisis occurred on the thirteenth day, certainly a rare event, and this after a course of intermittent temperature quite unlike what we are told to expect; or else two attacks of pneumonia followed one another, a rare event also, yet a supposition here rendered probable, as has been said, by the rate of pulse on admission, the development of physical signs, and the date of crisis. In either case how confused and doubtful are the materials for prognosis. At the outset who would venture to predict the future of either patient, or to put the chances of one above the other?

There are, however, features of difference between these patients

in the rust-coloured sputum, the stitch, and the definite history of exposure, which the one, B, had, and the other, A, had not. What, it may be asked, is the prognostic significance of such disparity? Everyone knows that characteristic sputum may be absent in the most typical pneumonia; the fact is variously accounted for, and teaches little as to the future. As for stitch, it is a symptom which, when severe, must always cause anxiety by preventing sleep. Last year we had an example of pleuritic pain in pneumonia, so acute and enduring (it was, as post-mortem examination showed, chiefly diaphragmatic) that of itself it seemed to determine the fatal issue.* There is no single element of treatment more important than to discover the means of easing such pain. But in the case in question, pleuritic stitch was not of this exceptional severity. Both stitch and rusty sputum made B's pneumonia more obvious than A's; they could hardly be said to make it either more or less threatening.

There were two points, however, in the comparison of the two cases, manifestly unfavourable to A, and favourable to B: one had reference to the extent of the local inflammation; the other, and, in my belief, the more important, had reference to its mode of origin. B's pneumonia obviously originated in chill. There was no such evidence in the case of A.

It need hardly be said that when pneumonia is found to spread from its first seat, and eventually to involve both lungs, the case is removed from the common rule, and the favourable prognosis which attaches generally to the disease must be withheld. Yet, for all that, spreading pneumonia is not necessarily severe, and it often comes abruptly to its term at or about the seventh day. In fact, it is not so much from anything we observe in the condition of these youths respectively that we are enabled to judge between them, it is from what we are told in reference to their antecedents. B got his pneumonia frankly as a consequence of cold; A got his, no one knows how or whence.

Once all idiopathic lobar pneumonia was attributed to chill. It is remarkable that in the present day a much enlarged view of its causation concurs with a more rigid and concise definition of its phenomena than that which formerly served. The observation of a multiplicity of causes has not led to any

* Post-mortem and Case Book, vol. v. p. 372.

parallel observation of varieties in clinical form. A pneumonia of chill is not separable by its symptoms from one that is pythogenic or alcoholic, or of some obscure nervous origin. We can easily imagine the several modifications which *ought* in the fitness of things, from a fireside view of pathology, to characterise these several forms; we do not as a fact observe them, or at the most we only observe them occasionally.

Such classification as we have is derived mainly from the seat of the lung inflammation, and it is always precarious and often misleading. It is true that apex pneumonia is apt to show active delirium, that spreading pneumonia is often severe, and double pneumonia on the whole of larger mortality than single, yet it is easy to insist too much upon such characters. Locality has no necessary connection with any particular form of attack; the extent of pulmonary inflammation is not always a measure of severity; and the fear that is often expressed, lest inflammation of an apex should end in phthisis, is altogether groundless. All that can be predicated is this, that a large number of antecedents, many and various causes, may eventuate in a temporary exsudation within the lung recognisable by a certain set of physical signs, little variable in themselves but of very uncertain significance.

Not less faulty and uncertain, to say nothing of its limited applicability, is the classification of pneumonia after death based upon the intimate character of the exsudation. The disseminated form of the disease, broncho or lobular pneumonia, differs indeed in many respects from the circumscribed or lobar (although the distinction may be little or none in particular instances), but the intimate histological characters are not always so different as to enable us from mere microscopic inspection, out of sight of the lung as a whole, to distinguish a so-called catarrhal from a so-called croupous pneumonia.

So then *the character*, and therein of course the prognosis of lung exsudation, is determinable not from its anatomical distribution, nor yet from its histology, even could that avail us in the patient's lifetime, but from what I must call provisionally the *genius* of the process itself, and this in its turn depends largely on the causes that actuate it.

That this is so, is in fact recognised amongst practical men, though the field of observation to which this view of the case

invites us has been almost neglected. Not what pneumonia is, but whence it comes is the main consideration. It is known, for example, to be specially dangerous with the subjects of renal disease or of alcoholism; there may be nothing out of the way in its proper symptoms, it is the evil association that gives to these a certain colour.

This insufficiency of pneumonia to speak for itself, this need of search beyond its own immediate phenomena to learn its precise meaning, may well suggest doubt as to the place that should be accorded to it in modern nosology. It is some test and evidence of progress in medicine that from time to time morbid conditions get transferred from the category of diseases to that of symptoms. Dropsy and diarrhœa and bronchitis were accounted diseases once, they are now symptoms of disease. In such light may we not reasonably place pneumonia? Pulmonary exsudation, we should then say, may be the result, near or remote, of a large number of conditions, some of them within sight, some not even thought about until lately. Not chill only, but alcohol, sewer gas, acute rheumatism, starvation, fatigue, and many other agencies which we are only beginning to recognise, are all of them in their several degrees provocative of this lung condition, and each carries its own measure of danger. That pneumonia, locally regarded, fulfils its course after a certain pattern, within a certain period, and with certain physical signs succeeding one another in a definite order, all this has been emphasised, and perhaps over-emphasised, in a generation not wholly freed from the teaching that made *le râle crepitant* the centre and pivot of the whole matter. But it has to be remembered as well that you may gauge the physical state of the pneumonic lung as precisely as you will, yet learn nothing of the nature of the disease of which it is but the partial expression. The fact is that the course of pneumonia appears to us far more uniform than it is, because physical signs, which are, and must be the same or similar in all cases, occupy so large a place in the description. The affection implies the sealing up of a portion of lung, and this process is indicated by crepitation, percussion dulness, tubular breathing, and so forth, signs which presently get modified as the exsudation liquefies. But place the physical part on one side, and where is the rest? Identity in physical signs concurs with the utmost variety in other respects.

The late Dr. Hughes Bennett hardly exaggerated when he wrote, "There may be pneumonia with none of its symptoms, and all its symptoms with no pneumonia." *

What we call by this partial and inadequate name pneumonia, is in reality made up of many factors, and when we know it better we shall be indifferent, so far as diagnosis is concerned, as to which of these puts in an appearance, or is the most prominent. Thus lobar exsudation goes practically always with pleuritis, often with herpes, sometimes with pericarditis; but any one of these will serve without the others. Pleural inflammation may have all the characters and associations of pneumonia with no inflammation of the lung †; pericarditis may precede or follow or accompany pneumonia; labial herpes not seldom shows itself with all the apparatus of pneumonic fever as to outset, duration, and pyrexia, while the lung itself escapes; nay more, as Andral told us more than sixty years ago, we may have notice in pulse, temperature, and respiration of an impending pneumonia, yet the storm never actually breaks either in the lung or any other district of the body.‡

It may sound like paradox, yet it is true that the same obser-

* *Practitioner*, vol. ii. p. 263. While these sheets are passing through the press, a woman of 38 comes under my charge in the second day of an illness that had commenced with severe rigor and headache. She was in a state of peculiar drowsiness, yet could be aroused so as to answer questions pertinently, but immediately after relapsed into stupor. She was not flushed; the breathing was not hurried; there was neither cough nor any morbid sounds whatever in the lungs. Her stillness and semi-coma seemed to point to some cerebral lesion, but there was no sort of palsy and no optic neuritis. Pulse was 100, temperature 103°.

On the fourth day of her illness, without other change, this patient exhibited herpes about the lips and chin; on the fifth day some small crepitus was audible over a limited area of the right base, temperature meanwhile varying between 103° and 104°, and the same drowsiness and lethargy being still the most prominent feature in the case. On the sixth day this woman had well-marked crisis; the same day redux crepitation was plainly audible, and on the seventh day the drowsiness had disappeared; the woman's manner was natural and her temperature normal.

During the first days of this patient's illness the hypothesis of pneumonia had been considered and rejected. In the end it could not be doubted that her condition corresponded best with the pattern of the disease we so name. Yet was it not a pneumonia of which the lung indications formed the smallest part?

† See Post-mortem and Case Book, vol. v. 371, where pleuritis was associated with cerebro-spinal meningitis.

‡ Andral, *Clinique Médicale*, case 12. See also Grisolle, *De la pneumonc*, p. 187.

vation which teaches us to distinguish one disease from another serves also to obscure their mutual relations and interdependence. We get more and more elaborate in assorting morbid states, but are slow and uncertain in associating them. Formal classification of itself distorts the natural face of nature, while it leaves altogether out of sight antecedent conditions of disease at that early stage of development, when their kindred and their common origin are unmistakable. What we want, and what in some of the substitutions of pneumonia we seem dimly to perceive, is a knowledge of morbid equivalents, such real insight, the fruit of exact observation, as is fancifully assumed to exist in the many shapes, "masked," "latent," "incipient," and the like, popularly ascribed to gout.

But to return to the question of prognosis. As yet the etiology of pneumonia has been too little studied to warrant any precise or complete statement as to its prognostic significance. Like acute arthritis, facial erysipelas, and other so-called chill diseases, unless it can be ascribed to that bodily exposure which is regarded as its proper cause, its origin is neglected and left out of sight. And not only so, but this compelling belief in the agency of chill leads us to assign that cause more often than the facts would warrant. This boy A, for example, "thinks he must have caught cold." He has no further reason for thinking so than a recollection of chilliness and a share in the popular belief that lung affections come in that way. Sensations of cold standing as they do at the beginning of all acute illness are easily mistaken for its cause, and we have to separate between actual chilling of the body, and the over-sensitiveness to cold natural to the febrile state. The etiology of the diseases in question, and especially of pneumonia and rheumatic arthritis, needs to be investigated afresh, more widely and with a more open mind.

I need not repeat here the provisional and incomplete conclusions of the Collective Investigation Committee upon this subject. It has always appeared to me, these conclusions apart, that such pneumonia as can be definitely traced to chill or exposure, is so far likely to run a favourable course; while, on the contrary, an obscure origin imports a doubt as to termination. Sometimes it will happen that the source of the attack is only partially known, and little can be gathered from such knowledge. Thus

pythogenic pneumonia, and that rare form of the affection that seems to be conveyed from person to person, will be very fatal in some instances, yet it is not necessarily of high mortality. Its danger varies with the particular poison of which it is the expression, as is the case with the specific fevers. There are instances of conveyed pneumonia wherein every individual attacked died, and instances where the deaths were few or none,* the variety of result depending obviously not on the lung condition, but on the nature of the poison underlying it. Again, there is, as I believe, a pneumonia of depression, so to call it, that, namely, which seems to be the issue, more or less direct, of nervous shock or injury (the lung inflammation following influenza may be one illustration of it), and which is preceded by nervous prostration without any discovered disease apart from the lung. It has for its initial cause sometimes over-fatigue, sometimes mental distress. I could quote several instances where pneumonia of such origin, although of small area, and judging from physical signs of no gravity, has been eventually fatal. In my view, therefore, it is the frank origin of B's pneumonia in a notable wetting that gives him the chief advantage in a forecast over A, who can give no account of the source of his illness.

To this less fortunate youth A let us now return for a moment. The last note of him, given above under date August 16th, would seem to leave little doubt that he had phthisis, nor can I say for certain, even now, that such is not the case. But this much is certain: that some parts of the lungs where phthisis threatened, and notably the apices, are now normal. At this time of writing (Sept. 23rd) the physical signs of consolidation are limited to the right base, where sharp, ominous crackles are still audible. The boy has hardly lost weight, notwithstanding his three months' illness and long-continued night pyrexia. On admission he weighed 6 st. 3 lbs., to-day he weighs 5 st. 12 lbs. 4 oz., the same, that is, as at the beginning of August. He gets about and takes walks abroad, but his pulse is still over 100, and he has troublesome paroxysmal cough, with but little, and that merely bronchitic sputum. He will soon be leaving for some convalescent hospital. That he will wholly recover is uncertain, but from his progress so far, and the history of like cases, I do not despair of it.

* See Collective Investigation Record, vol. ii. pp. 8 & 18.

But whatever the issue in this particular case, which, as we have seen, is exceptional in many respects, I feel warranted by former experience in saying this: when unilateral lobar pneumonia attacks a healthy individual after its usual manner yet fails o clear up as usual, you should hesitate long, and wait for unequivocal physical signs of excavation, before concluding that the patient has phthisis. Not seldom does it seem to be so; very rarely in my belief is it really so. Just as in B's case the fact of exposure causing the disease is favourable to its early and critical recovery, so in A's case the fact that chronic consolidation (or what passes for it) at one base is the sequel of acute pneumonia, is favourable to its ultimate recovery. I do not say that all such cases recover, or that none degenerate into phthisis. Neither statement would be true. I say only that with such physical signs the antecedent history is a favourable element in prognosis.

It is to this simulation of phthisis, especially in the young, that I would call especial attention. When pneumonic consolidation fails to resolve, when the exsudation is partly solid and partly fluid, and the crepitations are rendered sharp and penetrating owing to the extra conduction of sound by the sealed up portion of lung, the physical signs come to resemble very closely those of commencing phthisical softening. A thickened pleura (with sometimes, in addition, a small and physically unrecognisable empyema) makes this likeness the closer. Somehow or other, as I have repeatedly observed, this state of pleura so accentuates the auscultation signs, that both the extent and consistence of the lung consolidation are found after death to have been overestimated. In the later stages of acute hepatisation, on the eve, as presently appears, of crisis, we have all listened to the sounds I speak of with apprehension as to the integrity of the lung structure. Yet in a day or two these have disappeared with the rapid and complete recovery of the patient.

The fact that so few of these subjects of chronic pneumonia (as we commonly call it) die, that, as a rule, they pass out of sight well as to their general health, yet unrecovered so far as physical signs witness; in other words, the fact that this condition is far commoner in the wards, and especially in children's wards, than in the post-mortem room, makes it difficult to define the precise nature of the anatomical changes. My own experience would lead me to believe that *we habitually underrate the share*

taken by the pleura, and its large concern in the auscultatory phenomena. A tough and thickened pleura, sometimes enclosing an empyema too small in the circumstances to be discoverable by physical signs, and along with this a portion of lung not hepatised but carnified as by long pressure, such are the appearances, so far as I have seen, commonly revealed after death. That this carnified lung represents the seat of the original inflammation is the usual, but by no means necessary inference. It may be that it is that portion of lung which in the early stage was interposed between hepatisation and pleuritic effusion, and which, owing to the chronic nature of the process, had been so long compressed between the fluid and the unyielding lung as to become incapable of expansion. If that be so, it would seem to follow that the condition called chronic pneumonia is not always inevitable, inasmuch as an early and complete withdrawal of the fluid effusion would remove the injurious pressure in time to save the lung.

In conclusion, it may not be superfluous to notice that, as regards prognosis and the relationship of the two to phthisis, acute lobar pneumonia and acute bronchitis exhibit a marked contrast, while catarrhal or broncho-pneumonia, a combination of the two, occupies a varying position between them, and is sometimes nearly, sometimes remotely, associated with phthisical changes. Thus when acute bronchitis lingers, we begin to fear phthisis, and this fear increases when the morbid signs, ceasing to be general, are still audible at one apex. It is then too often that night sweats, wasting, and general failure combine to make the diagnosis of phthisis almost certain, notwithstanding that there is as yet no physical evidence of deposit, and in fact no more, so far as the ear can tell, than a localised bronchitis. The course of pneumonia reverses all this. When *it* lingers our fear of phthisis is not greatly aroused, and it is at the base rather than the apex that it usually stays. The fact that it is strictly localised and confined to one side is an element not of danger but of safety. The physical signs indicate a material change in the lung, and with but little alteration in this respect the patient goes on improving in general health until he is practically well.

The several points relating to pneumonia and its prognosis that have been insisted on in this paper may be summarised as follows:—

1. Exsudation into the lung, though it gives rise to definite physical signs following an orderly course, nevertheless arises under so many conditions that, like pulmonary œdema, pleural effusion, and acute arthritis, affections whose precise interpretation does not reside in their names, it should be regarded rather as a symptom than a substantive disease.

2. Admitting the general truth that lobar pneumonia in a healthy and temperate person commonly recovers, yet the indications to guide prognosis in particular cases are scanty and fallacious. Of such indications the mode of origin of the attack is amongst the most important though the least regarded; and, *ceteris paribus*, the more clearly the affection can be traced to the accident of exposure the more favourable is the outlook. The prognostic significance respectively of other modes of origin awaits further clinical observation.

3. Chronic lung consolidation, or such physical signs as are indistinguishable from it though dependent in large measure on the state of the pleura, are the less likely to end in phthisical disorganisation the more clearly it can be ascertained that the condition is the immediate sequel of unilateral lobar pneumonia.

GRANULAR AND TUBERCULAR CONJUNCTIVITIS.

By N. C. MACNAMARA.

Granular conjunctivitis, or ophthalmia. Synonyms: trachoma, follicular, Egyptian, military ophthalmia; papillary and vesicular conjunctivitis.

The fact of a disease having been described under so many different names is strong evidence as to the existence of vague ideas regarding its nature. In the case of granular conjunctivitis this diversity of opinion has arisen partly from the obscurity surrounding its pathology, and also from the loose way in which the term trachoma has been employed to designate one of the prominent features of this form of conjunctivitis. The word trachoma in works on ophthalmic surgery is frequently employed as synonymous with the term granular conjunctivitis; trachoma, however, simply means a rough or uneven appearance of the conjunctiva, and the surface of this mucous membrane may become rough as a result of various causes other than that which induces granular conjunctivitis. Nevertheless, the opaque, firm, fleshy elevations seen on the conjunctivæ of patients suffering from granular ophthalmia is almost peculiar to this affection, and if we restrict the term trachoma to this state of the eyelids there can be no harm in making use of the word; but we must clearly understand that trachoma, or the hypertrophied villous tissue of the conjunctiva, is the result of irritation of the structures implicated, and that this irritation likewise leads to the accumulation of lymph cells in the submucous tissues, and so to the growth of granules, the production of which have led to the term granular conjunctivitis, or ophthalmia.

In a considerable proportion of the out-patients attending our

ophthalmic hospitals we may see on everting the eyelids, after wiping away the moisture from their surface, that there are on the tarsal fold of the conjunctivæ a number of greyish pearl-like elevations, resembling a millet seed, or the eggs in the spawn of fish; their colour and transparency may be compared to that of catgut. These granules form in the sub-mucous tissue, and consist of an aggregation of round cells and a network of fine connective tissue; as the cells accumulate they press upon the surrounding structures and so produce a capsule. Granules of this description often extend over the orbital conjunctiva and into the cornea. Wherever situated, the granules sooner or later either disappear, or else undergo fatty degeneration, and in time their place becomes occupied by connective tissue, which gradually contracts and obliterates most of the blood-vessels and lymph canals, leading to the entire destruction of the mucous membrane. Changes of this kind in the conjunctiva produce alterations in the curvature of the tarsal cartilages, and so to ectropium, entropium, and trachiasis. In by far the majority of cases the growth of these granules is accompanied with the formation of opaque excrescences on the conjunctiva, due to hypertrophy of the villous structure of the mucous membrane. These fleshy projections resemble the florid granulations often noticed round a fistulous opening connected with diseased bone. The affection, however, which we are considering is not called granular conjunctivitis because of the granulation tissue above referred to, but on account of the granules which as a rule, although not necessarily, precede the increased growth of the villous structure of the conjunctiva.

The question naturally arises as to the cause of these granules and hypertrophied villi. This question has now been answered by the discovery of a specific micro-coccus in the affected tissues. From the researches of Sattler, Schmidt of St. Petersburgh, and many other observers, we learn that the trachoma-coccus closely resembles the coccus of chronic gonorrhœa. When introduced after cultivation under the conjunctiva of healthy animals, but more rapidly in half-starved creatures, this coccus causes the growth of the granules and hypertrophied villi of granular conjunctivitis. Cultivations of the trachoma-coccus are characteristic on blood serum and in agar-agar; they are invariably found in the granules and villous tissues of individuals or

animals affected with granular conjunctivitis. In fact, as in tubercular disease of the conjunctiva, we have distinct evidence of the presence of the tubercle bacillus in the affected tissues, so in granular conjunctivitis it has been as clearly demonstrated that the trachoma-coccus is present. We may further observe that as in tubercle the presence of the bacillus leads to necrosis of the tissues in which it finds a home, so the trachoma-coccus, if planted in the mucous membrane of the conjunctiva, produces changes which terminate in cicatrisation and contraction of the structures surrounding the micro-organism.

Staderini states* that if the trachoma-coccus is introduced beneath the epithelial layer of the conjunctiva, it is not until about the end of the second week that it causes any visible changes in the part. If sections of the tissues are then examined, granules, or the aggregation of white blood and lymph corpuscles, may be seen. As these cell-nests become matured they are permeated with a fine reticulum of connective tissue, and acquire an investing membrane formed in the manner above indicated. Subsequently hypertrophy of the papillary structure of the conjunctiva occurs, both the granules and overgrown papillæ being the result of irritation due to the presence of the trachoma-coccus in the tissues. It is the presence of this organism in the villi which renders them, and also the granules, so rebellious to treatment, and in this respect granular conjunctivitis differs from ordinary catarrhal affections of this mucous membrane.

The feature of granular conjunctivitis which, however, causes us the greatest anxiety is the vascular opacity of the cornea (pannus), so frequently met with among patients suffering from this form of disease, and which leads in so many cases to impairment of sight, if not to complete blindness. The first sign of pannus consists of a collection of lymph cells in the cornea beneath Bowman's membrane. Blood spaces and vessels form in this layer, and subsequently a tissue which possesses the properties of adenoid tissue. This new growth having a low degree of vitality, is apt to break down in small circumscribed areas, forming an ulcer, which as a rule, however, has a tendency to heal rather than to extend deeply into the cornea. In fact, typical nests of round cells such as those met with in the conjunctiva are also found in the cornea, and under the influence

* "Annali di Ottalmologia," Anno xvi., Fasc. 5, E. 6.

of the trachoma-coccus they give rise to very much the same changes in the structures involved.

The micro-organism I have so frequently referred to is obviously directly or indirectly a source of irritation of tissues in which it takes root, and the blood-vessels of the part are very apt to become hyperæmic in connection with the altered condition of nutrition thus induced. The engorged vessels lead to the formation of muco-purulent matter on the surface of the conjunctiva, and in this fluid a number of the spores, if not trachoma-cocci, are to be found. In this way we can understand how it is that the secretion from the eyes of persons suffering from granular conjunctivitis is infective, and so how it comes to pass that when once this form of ophthalmia occurs in a school or other such community it is likely to spread from one person to another.

How far hereditary tendencies modify tissues so as to render them susceptible to the attacks of certain micro-organisms we are unable to determine. But we know that persons of all ages existing in a dark, smoky atmosphere, such as that prevalent in the over-crowded parts of our large cities, are peculiarly susceptible to the inroads of micro-organisms; in addition to this, the conjunctivæ of persons living in filth and want are subjected to deleterious and irritating fumes or to mechanically suspended particles in the air, which are apt to excite an unhealthy state of this mucous membrane, and so to produce a favourable soil for the development of certain cocci. My friend Mr. Sydney Stephenson has lately given us a good illustration of the difficulty which exists in getting rid of granular conjunctivitis when once it has become established in a community such as that of a regiment, ship, or as in this instance, in a school (*Lancet,* April 5th, 1890). Mr. Stephenson observes that in the Central London District School at Hanwell ophthalmia has always existed; and had become so intractable and so great a bane to the institution that in 1889 the authorities determined to carry out a complete and well-devised system of isolation, or rather strict separation of children affected with granular lids from the rest of the community. Iron buildings were erected at some distance from the school, every attention being paid to the water supply, drainage, and cleanliness of these houses, into which children were sent if suffering from symptoms of ophthalmia.

Having been admitted into the isolation school, the inmates are taught, fed, and live absolutely apart from their fellow-creatures, and their conjunctivæ are carefully treated by Mr. Stephenson. The result so far has been satisfactory, and although, in consequence of the flow of children from the slums of London into the school, fresh importation of the disease occurs, nevertheless, under a system of careful medical supervision and isolation, such as that now in force in the Hanwell District School, there is every reason to believe that granular ophthalmia will disappear from among the inmates of this establishment.

SYMPTOMS.—As I have before remarked, if we examine the conjunctivæ of a number of children, or it may be of older people, living in the poorer quarters of London, or other large cities, especially among the lower classes of our Irish and Jewish population, we shall find, although they may not complain of having anything the matter with their eyes, that the following condition exists. On the palpebral conjunctiva, especially in the tarsal portion, a number of small, light grey or yellow, somewhat transparent, raised granules may be seen; these are sometimes so minute that a lens and oblique illumination is necessary to enable us to perceive them. In conjunction with these granules the surface of the tarsal conjunctiva is redder than natural, unless the patient happens to be anæmic; the papillæ on its surface are swollen and greyish; the retrotarsal fold projects considerably, especially on the everted upper lid. We may notice patches of the enlarged papillæ elevated above the surrounding conjunctiva, and it is usual in such cases to find distinct traces of mucus in the conjunctival sac. It is possible to mistake the granules above referred to for the accumulation of lymph-like cells forming a row of vesicles on the retrotarsal fold of the lower lid, and which are frequently seen in cases of simple catarrh, or in persons who overwork their eyes. The chalky white deposits in follicles of the meibomian glands of old people have been mistaken for the granules above referred to; and the patches of swollen papillæ, for those which form round a foreign body or the perforation of a chalazion.

This passive and early stage of granular conjunctivitis may continue for months or years without giving rise to any obvious symptoms or inconvenience, spontaneous recovery, however,

probably never occurs; but if the individual is removed to favourable surroundings, and is well-fed and properly treated for some months, the granules may entirely disappear. In exceptional cases the granules commence in the orbital conjunctiva and cornea, at any rate we find them in this situation and not on the palpebral mucous membrane.

In by far the majority of cases the first indication of disease in the affected eye is an attack of conjunctivitis; the patient comes to us suffering from intolerance of light, lacrymation, a sensation of grit in the eyes, and it may be pain; there is also a muco-purulent discharge, and the eyelids stick together in the morning. These symptoms are aggravated by exposure to light, or by any attempt to use the eyes. On everting the lids we may see well-marked granules on the tarsal conjunctiva, especially of the upper lid, the papillæ are engorged, often in patches, and the vessels of the conjunctiva throughout the whole of their extent are much injected; flakes of mucus may usually be observed on the tarso-orbital fold of the conjunctiva and about the inner canthus. An attack of this kind may pass off in the course of two or three weeks if properly treated, and the patient then returns to his employment, or, if in the army or navy, to duty. Men affected with granular lids are, however, liable to an attack of severe conjunctivitis at any moment if exposed to unusual sources of irritation, such as the dust of a tropical country, excess of heat, or cold and damp. Among the civil community the same and various other atmospherical impurities start a fresh attack of inflammation of the conjunctiva; people affected with this condition of the eyelids are constantly suffering from "inflamed eyes," which render them unfit to carry on the work of their calling.

Every fresh outburst, or continued conjunctivitis, leaves the mucous membrane in a worse condition than it was previously; after the acute symptoms have passed away the granules may become indistinct, if not altogether covered over by the infiltrated sub-conjunctival structures; at the same time the hypertrophied papillæ stand out more prominently upon the surface of the mucous membrane. Subsequently, or it may be from the commencement of the disease, the granules appear on the orbital conjunctiva and in the cornea; in the latter structure they may be seen as opaque cream-coloured spots or patches, surrounded

by a hazy area of corneal tissue. This condition indicates the commencement of pannus, the cornea becoming first affected in its upper portion, where it gradually assumes a rough opaque condition (aptly compared to that of ground glass), into which numerous dark red blood-vessels pass; these vessels seem to project above the surface of the cornea. The extension of pannus in the cornea is generally a slow and painless process, but in some cases it is accompanied with marked photophobia, lacrymation, and considerable pain.

It is impossible to predict how long it may take for the disease to run its course, or the cornea become so opaque as to prevent the patient from gaining his living; but it would almost seem that so long as any portion of the conjunctiva, or the anterior layers of the cornea, exists the granules continue to grow in the part, doubtless produced by the irritation caused by the trachomacoccus in the connective tissue of the affected structures. As the disease becomes chronic further alterations in the eyelids take place; in some of these cases there is no difficulty in everting the lids, which are nevertheless obviously much thickened, and their surface covered with hypertrophied papillæ; these may have run into one another and formed a tough vascular structure, apt to bleed if roughly handled. In this condition the lids cannot close properly over the eyeball, the puncta are everted, and ectropian frequently supervenes. In other patients we find that it is difficult to evert the upper eyelids in consequence of the growth and contraction of the connective tissue which has been formed in the part during the more acute stages of the disease. The palpebral fissure is narrow vertically, from the abundant production of cicatricial tissue in the neighbourhood of the entrance of the palpebral arteries. In these cases entropium often follows, and the incurved cilia add greatly to the distress which the patient suffers.

Changes such as those referred to necessarily displace or choke the meibomian glands, their ducts become obliterated, and the retained secretion may become inspissated and calcified, leading to the chalk-like nodules so often seen in chronic cases, not only of granular but also in other forms of conjunctivitis. Another condition often met with in these long-standing cases is the adhesions which form between the inner surface of the lids and the eyeball. These fleshy bands of adhesion are noticed when

the lower lids are everted, and the eyeball is then directed upwards; they are formed of cicatricial tissue, and connect the surfaces formerly covered by the tarsal and orbital conjunctiva, and as the secreting structure of the latter has been completely destroyed the adhesions look dry and are sometimes covered with bran-like scales. Alternately, from partial or complete destruction of the normal elements of the cornea, it may yield to the intraocular pressure, and a projection in the form of a staphyloma results. Well might some surgeons have described this affection of the eyes malignant ophthalmia, for when once established it spreads from one texture to another, involving destruction in each and all the parts implicated, and it is almost as irremediable when once it has taken root in the conjunctiva as any form of malignant disease in the tissues which it invades.

Acute Granular Conjunctivitis.

We occasionally meet with cases of granular conjunctivitis in which the disease runs a rapid course. In instances of this description the granules appear almost simultaneously upon the conjunctiva and cornea, producing extensive infiltration of the tissues. The eyelids, in the course of a week or so, become greatly swollen and thickened, the tarso-orbital folds being completely occupied with fleshy-looking granulations. The cornea is hazy, and a number of well-defined granules may be seen in its substance; pannus forms rapidly, and may extend in breadth several millimeters in a single night. The secretion from the surface of the conjunctiva is profuse, and the patient suffers from great pain in his eyes, and from general feverishness. The prognosis in cases of this description is unfavourable, for although it may be possible to relieve the acute symptoms the cornea seldom regains its natural transparency. In these acute cases the granules are rapidly covered in by the infiltrated conjunctiva, and it is then more from the condition of the cornea than of the eyelids that we arrive at a diagnosis as to the nature of the affection from which the patient suffers. In the most favourable circumstances the attack rarely subsides within a month or six weeks from its commencement, and not unfrequently passes on into a chronic state, the granules reappearing on the conjunctiva as the swelling

in this part diminishes. The granules in the cornea are often so numerous that they are apt to become confluent and then break down, producing rather deep ulcers, and sometimes abscesses. The acute symptoms having passed away, as I before remarked, the cornea is almost always permanently damaged to a greater or less extent, it may, however, continue to clear slowly for some months after the active disturbance has subsided.

Treatment.—From the account I have given of granular conjunctivitis it is obvious how important it is to recognise the presence of granules in the conjunctiva at an early stage of their growth. In schools, such as the one to which I have referred, it is impossible to over-estimate the advantages to be derived from the examination and isolation of children by a skilled surgeon before admission into such an institution, so as to weed out those affected with granular lids. The treatment of cases, whether in schools, regiments, families, or any other communities, should in the early stages of the disease be conducted on the principle that granular conjunctivitis is the work of a vegetable micro-organism planted in the affected structures, and that this coccus must be destroyed if the disease is to be overcome; further, the trachoma-coccus may spread from one person to another through the discharge from an eye affected with the disease. This contagious property of the discharges from the eyes of an affected person is retained for any length of time if the matter is kept in a dry condition, as, for instance, on dry linen, and the more acute the attack the greater is the infective power of the matter formed on the surface of the conjunctiva. There seems good reason for supposing that the susceptibility of the conjunctiva to receive and form a favourable soil in which the trachoma-coccus may develop, depends on the healthiness or otherwise of the mucous membrane at the time the coccus reaches it, and also to some extent on the individual's general state of nutrition. Certain chemical substances have the property of exciting the rapid growth of this micro-organism, as, for instance, atropine, which, if frequently applied to the eyes of a person affected with granular lids, may produce an acute attack of conjunctivitis.

Supposing we detect granules and patches of hypertrophied papillæ on the conjunctiva of an individual who has perhaps

consulted us on account of some slight irritation in his eyes; our first care, if possible, should be to guard those brought in contact with the patient from infection. This remark applies with equal force to the case of children on admission into a school, although they may not be aware of having anything the matter with their eyes. The basins, towels, and everything likely to be contaminated in any way by the secretion from the conjunctiva of such a person must be carefully disinfected and guarded; at the same time the food and hygienic condition of the individual should be strictly attended to. Beyond this, in the early stages of the disease we may do much to weaken if not to destroy the organism on which it depends. I think the best local application for this purpose is the chloride of zinc. The patient's eyelids having been everted should be wiped dry, and then painted over with a solution of chloride of zinc (2 grains to an ounce of water); this should be repeated every morning, and a solution of perchloride of mercury (1 part to 15,000 of water) dropped into the eye three times a day, and the eyelids also bathed with this lotion. Before going to bed at night an ointment (hyd. oxyd. flav. gr. viii. iij, iodoform gr. iij, vaseline ℥ss) should be freely applied to the margin of the eyelids. Treatment of this kind must be employed until all traces of the granules have disappeared from the eyelids, and even then the individual's eyes should be examined once a week so as to destroy any fresh growth of the disease.

Unless, however, in the case of military or naval surgeons, or those in charge of schools and so on, it is seldom in practice that we meet with cases of granular conjunctivitis in its primary stage. As a rule we are consulted by patients who complain of having had "weak eyes" for a considerable time, their condition varying between irritation and inability to use the eye in consequence of more severe attacks of ophthalmia. On everting the lids we find indications of granular conjunctivitis with mucopurulent discharge from the eyes, and if pannus has commenced beneath the upper eyelid with considerable photophobia, and probably pain in the eyes. In cases of this description equal parts of solution of perchloride of mercury (1 to 14,000) and a solution of cocaine (4 per cent.) should be dropped into the eyes three times a day; iced compresses are a great relief if applied over the eyelids for quarter of an hour at a time, three

or four times a day. At bed-time an ointment may be smeared along the ciliary border of the eyelids (hyd. oxyd. flav. gr. viii. iij, iodoform gr. v, vaseline ℥ss), and the ung. belladonna be smeared over the eyebrows and temple, it can be washed off in the morning. The eyes should be shaded with dark neutral-tinted glasses, but the patient allowed to go out as much as possible in the open air. Treatment of this description will usually alleviate the more acute symptoms; and subsequently after the use of a solution of cocaine the everted lids may be freely dusted every morning with a fine powder composed of equal parts of tannic acid and iodoform. In some cases, if this plan does not succeed in producing absorption of the exudation and hypertrophied papillary structure, a crystal of sulphate of copper may be applied to the lids every other day, a solution of cocaine having first been dropped into the eyes twice or three times to relieve the pain produced by the application of the sulphate of copper. The length of time such treatment is to be continued must vary with the condition of the conjunctiva. If in the more chronic cases we find that the hypertrophied papillæ form prominent patches, as they often do, surrounded by comparatively healthy conjunctiva, we may, after the application of a solution of cocaine, shave off, or, I think still better, scrape away the enlarged papillæ, and after the bleeding has ceased paint the raw surface over with a 3 per cent. solution of chloride of zinc, subsequently using the solution of mercury and iodoform ointment as above directed. The electro-cautery may be applied with advantage to single granules or to patches of hypertrophied papillæ, doubtless containing granules; but I think the careful and, if possible, entire removal of the affected tissues, and then the application of the solution of chloride of zinc, or of a crystal of sulphate of copper, more effectually destroys the organisms of granular conjunctivitis than any other means of treatment with which I am acquainted. If the hypertrophied papillæ are very abundant and widely spread over the conjunctiva we may be disposed to try in the first instance the effect of scarifying the part freely, and subsequently dusting the tissue over with the tannic acid and iodoform powder.

With reference to the treatment of pannus of the cornea, if slight, it will clear away under treatment such as that recommended for the relief of the conjunctivitis, the mercurial and

iodoform ointment being applied to the margin of the lids, and also smeared over the everted upper lid before the patient retires to rest for the night. The mercurial lotion must also be used. In the more severe cases of pannus it has been proposed to inoculate the conjunctiva with gonorrhœal matter so as to excite acute inflammation in the eye, and thereby the reabsorption of the exudation and organised tissue in the cornea. An infusion of jequirity has been used with a certain amount of success to excite inflammation in the eyes of persons suffering from severe pannus. A weak infusion of the seeds of jequirity is to be rubbed over the inverted eyelids. A single application is sufficient to cause a severe ophthalmia in most cases, but if it does not produce this effect the infusion may be applied a second and a third time. The obvious objection to the employment of means of this kind is that you cannot by any possible treatment always control the inflammation of the eyes when once it has been excited, and in attempting to cure the pannus the cornea and the eye may be destroyed. The same objection applies to cauterisation of the pannous cornea. My experience has not led me to form a favourable opinion regarding the plan of excising a zone of two or three mm. in width from the circumcorneal conjunctiva in cases of pannus; such treatment is seldom followed by any permanent relief in cases of vascular opacity of the cornea.

Operations for the cure of entropium or ectropium should seldom be resorted to for the relief of malposition of the eyelids due to granular lids, at any rate not until either the reabsorption or transformation of the exudation into the connective tissue has advanced so far that no considerable contraction is to be apprehended after the operation.

With reference to the treatment of acute granular conjunctivitis we should rely chiefly on the constant application of iced compresses to the eyelids, and the instillation of a solution of perchloride of mercury, 1 to 6,000, with the iodoform and belladonna ointments, to the eyelids and the skin on the temple at bed-time. If only one eye is involved the other eye should be protected by means of a watch glass properly fixed so as to preserve the sound eye from contamination with the matter formed on the diseased conjunctiva. The patient's general health must be attended to, and strict attention paid to dis-

infecting any articles of linen he may have used, so as to prevent the spread of the disease from one person to another.

Tubercular Conjunctivitis.

We occasionally meet with cases of tubercular affection of the conjunctiva, and in such patients portions of the diseased structures have been removed and tubercle bacilli clearly demonstrated in the part. Tubercles develop in the eyelids as in other parts of the body either as a primary affection, but more frequently it appears to extend from the mucous membrane of the nares through the nasal duct and sac to the conjunctiva, appearing first near the puncta. Whether the disease is primary or secondary, it produces in the first instance granules of the conjunctiva and hypertrophy of the papillæ similar in appearance to those met with in cases of granular conjunctivitis. The swollen villi are perhaps more abundant in tubercular disease, and they cover the whole surface of the conjunctiva, being more vascular, or at any rate more easily made to bleed, than is usually the case in granular conjunctivitis. The bacillus of tubercle, like the trachoma-coccus, evidently finds a soil in the conjunctiva favourable for its growth, and by exciting irritation in the part it leads to the formation of granules and hypertrophy of the papillæ similar to that described in trachoma. There is this difference, however, between the two: tubercular affections of the conjunctiva almost always lead to ulceration, whereas the tendency of trachoma is to produce a growth of cicatricial tissue and its consequences.

In most of the recorded cases of tuberculosis of the conjunctiva there has been enlargement of the lymphatic glands in front of the ear and below the jaw on the affected side; it is remarkable that as a rule only one eye is involved in the disease, whereas in granular conjunctivitis both eyes are generally affected at the same time. Pannus usually appears in the cornea from an early stage of tubercular conjunctivitis.

On everting the eyelids of a person suffering from tubercular disease of the conjunctiva, in its early stages, we notice prominent granules on the mucous membrane, and a number of florid swollen papillæ. The enlarged papillæ soon cover the whole of the tarsal conjunctiva, and become flattened against the

surface of the eyeball; between these papillæ there are furrows often filled with muco-purulent matter. As the disease progresses the surface of the swollen papillæ may ulcerate, and so extensive do these ulcers become that they have been mistaken for rodent ulcers, or for lupus, epithelioma, and for syphilitic disease.

The treatment of tubercular disease of the conjunctiva must be based on the fact that this affection is a constitutional one as well as local, and that hygienic precautions are as necessary in the way of pure air, water, and food as local treatment. Regarding the latter, I think that the diseased tissues should be completely scraped away, and when the bleeding has ceased the part painted over with a solution of chloride of zinc, and subsequently covered with the iodoform and tannic acid powder. The solution of perchloride of mercury should be employed as described in the treatment of granular conjunctivitis.

THE CAUSES AND TREATMENT OF ASTHMA.*

By Dr. DE HAVILLAND HALL.

In order that it may be clearly understood what I include under the term asthma I will quote Hyde Salter's classical definition, which runs as follows: "Dyspnœa of peculiar urgency and violence, generally paroxysmal and recurrent, often periodic, not necessarily attended by cough or expectoration, accompanied usually by dry râles, and compatible with easy and healthy respiration in the intervals of the attacks." It will be at once seen that here we have to do with a definition founded purely on clinical data, it is therefore one to which all can give their adhesion, no matter what may be their views as to the immediate cause of the paroxysm. As the foundation of all rational treatment is based upon a clear comprehension of the pathological conditions underlying the disease, it will be well to allude to the theories which have been adduced to explain the curious set of symptoms occurring during an asthmatic paroxysm. At the present time we need consider only three theories, viz.:—

1. Bronchial spasm.
2. Vaso-motor neurosis.
3. Tonic spasm of the diaphragm.

Practically the dispute is now limited to the advocates of the first two theories, tonic spasm of the diaphragm not having received much support of late. The oldest theory, and the one which has secured the greatest number of supporters, is that which attributes the asthmatic paroxysm to a spasm of the

* A Paper read before the East London and South Essex District of the Metropolitan Counties Branch of the British Medical Association, March 20th, 1890.

bronchial tubes, brought about by a contraction of the muscular tissue contained in their walls. As Professor Fraser has well pointed out, a very important fact in favour of the view of bronchial spasm is the circumstance that the nitrites, which have so marked an action in relieving the dyspnœa of asthma, are known to lessen the contraction of non-striped muscle, whereas by dilating the blood-vessels they produce hyperæmia, and would therefore increase the obstruction in the tubes if this were due to vaso-motor influence. A careful consideration of the whole subject has convinced me that at the present time the weight of evidence is in favour of the bronchial spasm theory.

The second theory supposes "a tumefaction of the bronchial mucous membrane in consequence of dilatation of its blood-vessels through vaso-motor influence."* This view is supported by the fact that many asthmatics complain of a sense of obstruction in the nose at the onset of a paroxysm, and that this obstruction sometimes continues during the whole period of the attack. Moreover, a rhinoscopic examination shows that the mucous membrane is actually swollen. That there is also an exudative element is shown by the symptoms of a catarrhal nature which attend the attack, and by the profuse expectoration which occasionally accompanies the termination of it.

Sir Andrew Clark suggests that asthma may be closely allied to urticaria, and that a condition resembling the wheals met with in this disease occurs in the bronchi.

May it not be possible that the theory of bronchial spasm and the theory of vaso-motor neurosis may both truly explain the phenomena of asthma, and that though in the majority of cases the contraction of the lumen of the tubes is due to spasm of the bronchial muscles, nevertheless, in a certain number of cases, this diminution in the lumen of the tubes is brought about by swelling of the mucous membrane from vaso-motor paralysis? Nay, I might go even a step further and say, that in the same case spasm of the bronchial muscles might co-exist with a tumefaction of the mucous membrane, both the spasm and the hyperæmia being the common effects of the irritation of the nerves of sensation.

The third theory—tonic spasm of the diaphragm—offers so inadequate an explanation for the symptoms observed during an

* Ziemssen's System of Medicine, vol. iv. p. 549.

attack of asthma, that it is unnecessary to do more than mention it.

Before leaving the subject of the pathology of asthma, I must say a word about the nasal origin of the disease, of which Dr. Bosworth,* of New York, is so ardent a champion. He makes the startling assertion that a large majority, if not all cases of asthma, or vaso-motor bronchitis, as he calls it, are dependent upon some obstructive lesion in the nasal cavity. He describes 46 cases of asthma, in all of which he found marked intra-nasal changes, and which were treated locally. Of the 46 cases thus treated, 28 were cured, 12 improved, 1 unimproved, and 5 unknown. Hack† has reported a case in which an attack of bronchial asthma was produced by the application of a powerful irritant (galvanic cautery) to the nasal mucous membrane, in a person who had previously never suffered from the complaint.

Schnitzler‡ records a case of asthma with universal urticaria, both removed through the healing of a chronic rhinitis. E. Fraenkel, Schech, Sommerbrodt, and many other writers, have also reported cases in which treatment directed to the nasal cavities, as, for instance, the removal of polypi, the rectification of the septum, and the destruction of hypertrophies of the nasal mucous membrane, has brought about a cessation of the attacks of asthma. Without, therefore, going to the absurd lengths to which Bosworth's enthusiasm has carried him, we may admit that a certain proportion of the cases of asthma is dependent on pathological changes in the nose and adjacent parts, and that a suitable treatment of these conditions is an important element in the cure of the disease. I was struck on looking back at my case-book to see the frequent mention of intra-nasal disease in cases of asthma before I had any idea that a causal relationship existed between the two. The practical outcome of this is that a careful rhinoscopic examination should be made in all cases of asthma. The view that I am myself inclined to favour is that asthma should not be regarded as a distinct disease, but merely as a symptom. Like many other disturbances of the respiration, as for instance cough and dyspnœa, asthma does not point to any one particular anatomical lesion, but is common to a very

* Diseases of the Nose and Throat, p. 244.

† Brit. Med. Journal, 1886, vol. i. p. 247.

‡ Centralblatt für Laryngologie, vol. ii. p. 9.

large number of pathological conditions. The adoption of this view will facilitate the treatment of the disease, in that though the symptom receives its due attention, the physician will have to look beyond it for the cause, just as no medical man of intelligence would be content to treat a cough without understanding its cause. All systems of classification in our present state of knowledge, and in view of the recent rapid development of medicine, must necessarily be of a temporary nature, and the only purpose which they can reasonably be expected to subserve is to facilitate the arrangement of cases and the investigation of their origin. However carefully or elaborately the plan may be drawn up, there are certain to be cases which it will be difficult to assign to any particular division, and others again which might be placed under more than one head. The simplest classification of the varieties of asthma seems to me to be the following, which is much the same as that employed by Dr. Thorowgood,* and by Dr. Hyde Salter † :—

I. Primary or Idiopathic.—This will embrace only a few cases in which the disease seems to be a true neurosis; later investigations, however, may tend to show that even these cases ought to have been assigned to one of the groups in the second division.

II. Secondary—

1. Hæmic.—Where the spasm is brought about by the circulation of impure blood, as in gout, rheumatism, and other diathetic affections.
2. Reflex.—As from the nose, possibly also from the irritation of a loaded stomach, or from the uterus, &c.
3. Where the spasm complicates bronchitis, emphysema, and other chest affections.

In turning our attention to the etiology of the disease it is important that we should discriminate between the causes of the *disease* and the causes of the *paroxysms*. Thus as Hyde Salter‡ has pointed out, the two are sometimes the same, that which has laid the foundation of the disease will produce the subsequent attack—*e.g.*, bronchitis may be the cause of the disease, and any subsequent attack of bronchitis may bring on a

* Notes on Asthma, 2nd edition, p. 15.

† Reynolds' System of Medicine, vol. iii. p. 512.

‡ Lancet, vol. ii., 1866, p. 259.

paroxysm of asthma. Ipecacuanha may produce the paroxysm, but could not produce the asthmatic tendency. All the evidence which has been collected is in favour of the view that the cause of asthma lies in an increased irritability of the mucous membrane of the respiratory track, including of course the mucous membrane of the nose and naso-pharynx. Hence the frequency with which the first attack may be traced back to measles, whooping-cough, or bronchitis occurring in early life. A point which comes out very strongly, and one which is quite contrary to the opinion of the general public, is the fact that a larger number of cases take their commencement in the first ten years of life than in any subsequent equal period.

Heredity seems to play an important *rôle* in asthma; Lazarus* states that in 15 per cent. of the cases it is hereditary.

Treatment.—I take it that in the vast majority of cases the patient consults his medical attendant in one of the intervals between the attacks, and it will therefore be convenient to direct attention to the treatment in the intervals, and then proceed to make some remarks on the measures to be taken in order to cut short the attack.

Bearing in mind the view already expressed, viz., that asthma is to be looked upon as a symptom rather than a disease, it is most important that the patient should be surveyed from all points of view, that his past history should be inquired into, the date of the commencement of the attacks, the antecedent disease, and his family history. Then a careful physical examination should be made, and any weak points in his constitution carefully noted. I lay great stress on the weight, especially in a chest complaint like asthma. Most asthmatics are below the average weight, therefore they can usually ill afford any loss, so that a treatment which still further reduces their weight should be regarded with suspicion. On the other hand a steady weight, or a slight increase in weight, may be considered of favourable omen. In regard to general treatment, first and foremost comes *diet.* As a rule asthmatics soon find out for themselves the diet that suits them, and they learn to avoid certain articles which tend to bring on attacks. Still there are some rules which are of more or less universal acceptation. To begin with, the asthmatic must recognise that the so-called pleasures of the table are not

* Centralblatt für Laryngologie, vol. iv. p. 87.

for him. He must live plainly and eat moderately, in fact he should learn to rise from the table feeling that he could still make a meal. The meals should be distributed at regular intervals, and the patient should take nearly the same amount of food at each. Made dishes and pastry are to be avoided. Meat is to be eaten with moderation, more especially if the patient is gouty or liable to hepatic derangements. Fluids should be taken at the end of the meal, and a tumblerful of hot water taken the last thing at night will lessen the need for drinking at meal times. Supper should be avoided, the last meal being taken not later than seven, and it should be the lightest of the three principal meals. As regards the question of stimulants, this depends mainly on two points—one positive and one negative; if digestion is improved by the addition of a little alcohol with the meal the patient may take some, usually a glass of claret, burgundy, or dry sherry, or a glass of weak whisky or brandy and water. On the other hand, the existence of lithiasis contraindicates, as a rule, the employment of alcohol.

Next to diet comes exercise, and in respect to this it may be stated that the asthmatic is benefited by daily regular exercise; this, by increasing the oxygenation of the blood, tends to keep up the vigour of the system.

The sleeping room should be well ventilated, and the bed a mattress or a spring bed, and the hours early.

The choice of a locality for residence is curiously affected by the idiosyncrasy of the individual. Attempts have been made by some writers to minimise this influence, but on the day while I was writing this an old patient of mine, a victim to asthma, came in and gave the following account of himself. He stated that for fourteen years he has suffered from difficulty of breathing at night whenever he slept out of London, but that he has never had an attack in London. In the summer of 1888 he went to Margate on a Sunday, the same night he had a very severe attack of asthma which lasted nearly all the next day. Monday night, however, he slept well, and Tuesday he was free from shortness of breath, but at night he had such a fearful attack that he left Margate the next morning, and when half-way to London his breathing became easy.

This and many similar cases have convinced me that locality does exert a potent influence on the onset of asthma. The only

general rule that can at all be formulated on the subject is that the air of London and other towns seems to be more acceptable to the asthmatic than the most exhilarating air of the country and seaside.

After attention to these general rules comes the inquiry into any particular predisposing cause on the part of the patient himself. And here I will first speak about the treatment of nasal affections as the evidence of the part played by them in the production of asthma is too great to be disregarded. I would, therefore, urge you as a matter of routine to carefully investigate the condition of the nose and adjacent parts in every case of asthma. Though I cannot agree with Bosworth* in his astounding statement, "that in the very large majority of instances an asthma is dependent primarily upon a diseased condition of the nasal mucous membrane," still I am convinced that benefit will result in most cases if the nasal affection be properly treated. In the *Lancet* for June 15th, 1889, I described the measures I advocate in the treatment of hay asthma, and precisely similar treatment is required for ordinary asthma. Indeed I am not prepared to admit that there is any essential difference between the two diseases. In addition to the nose, the naso-pharyngeal cavity and the pharynx should be investigated, and adenoid vegetations and enlarged tonsils removed if present. Having then disposed of the upper part of the respiratory tract any abnormal condition of the lungs should demand our attention. Bronchitis and emphysema are, of course, the two most common. Digestive disturbances require to be treated in the usual way, especial care being taken to ensure a daily action of the bowels, and if the liver is inactive an occasional dose of calomel or blue pill, or euonymin in combination with the compound rhubarb pill will be found useful. Uterine and other visceral disorders may also require attention. Gouty and rheumatic conditions will be benefited by alkalies. It has been pointed out that a skin disease may co-exist with asthma, and this is not a mere coincidence, but the two are to be regarded as the result of a common cause, hence the appearance of the skin affection may suggest a suitable therapeutic procedure. In this connection arsenic naturally suggests itself, and it is one of the few drugs which have any effect for good when taken in the intervals between the

* Diseases of the Nose and Throat, p. v.

attacks of asthma. It is now well recognised as a potent means of promoting blood formation, and is therefore largely employed in cases of anæmia. Many asthmatics are poorly nourished, and in these cases arsenic is worthy of a trial as well as when skin affections exist. I usually give it in the form of a pill containing $\frac{1}{8}$th grain of arseniate of iron twice or thrice daily after meals. The only other drug which seems to me to have any special effect on asthma is the iodide of potassium, and I was much surprised to find that two of the leading text-books of the day, viz., Bristowe and Roberts, do not mention it. On the other hand, Tanner,* whose work is full of practical suggestions for treatment, says that it "certainly works wonders," and that there is "no single remedy which can compete with it." Fagge has also found it very serviceable in some cases. With these statements I cordially agree. I have had hospital patients taking the drug continuously for months, and coming again after an interval and asking for some more of the old medicine as it did them so much good before. Dr. C. J. B. Williams† gave a mixture of 2 or 3 grains of iodide of potassium and 10 to 15 of the bicarbonate of potassium with strammonium or belladonna, and says, "with a combination of this kind I have cured or greatly relieved hundreds of cases of asthma."

Professor Germain Sée‡ uses iodide of potassium not only for the mere relief of the paroxysm but as a means of effecting the cure of the disease itself, and regards it as the most reliable agent in the treatment of asthma. On the other hand, Hyde Salter§ said that iodide of potassium "does not deserve so high a place as has been given to it." I usually give it in 5 grain doses in combination with half a drachm of aromatic spirits of ammonia three times a day.

Of the employment of galvanism and of the use of compressed and rarefied air I have no experience, so I shall merely remark that in the latter plan of treatment the patient is made to inspire compressed air and to expire into rarefied air. This plan cannot be regarded as a cure, it merely allows time to discover and treat the cause of the asthma. It also diminishes the amount of

* Practice of Medicine, 7th ed., vol. i. p. 600.

† Medical Times, vol. i., 1882, p. 651.

‡ *Ibid.*, vol. i., 1878, p. 149.

§ Lancet, vol. i., 1864, p. 92.

emphysema and improves the general condition of the patient by enabling him to take more exercise.

When we come to consider the various forms of treatment which have been proposed for the relief of the asthmatic paroxysm we are fairly bewildered by the innumerable suggestions that have been put forward, so that here it must suffice if I mention some of the most important.

It will simplify matters if I group the medicinal substances under the three heads of drugs administered by inhalation, those given subcutaneously, and those by the mouth. First of all then, for inhalations. I suppose the most universally administered inhalation is nitre paper, and in my experience it is rare to find a patient who has not tried it, and who, having tried it, has not derived a certain amount of benefit from it. In some cases, indeed, it acts almost like a charm. No sooner does the patient inhale the fumes than the spasm relaxes, and he may even drop off to sleep holding the apparatus in his hand. It is desirable to have the paper prepared in a uniform manner. The formula in the Westminster Hospital Pharmacopœia for making it is to saturate white blotting paper in a solution of nitrate of potassium, 45 grains to the ounce, and when dry to sprinkle a few drops of tinct. benzoin co. over the paper. The addition of the tincture of benzoin makes the inhalation more agreeable. How nitre paper acts I do not pretend to say, nor can I give any rules for its administration. As it is a perfectly harmless and an easily employed method of treatment I always suggest its use at an early period. A patient of mine pointed out to me a very simple plan of inhalation. He places a saucer in an ordinary chimney-pot hat, the nitre paper is then lighted and thrown on the saucer, the patient holding his head over the hat, and if he desires to get the fumes more concentrated he can place a towel over his head.

Himrod's cure is a well-known nostrum for the relief of asthma, and I know patients who never think of leaving home without taking it with them. The following has been suggested as its probable composition, and I have found it very efficacious :—Equal parts of powdered lobelia, strammonium leaves, black tea, and nitrate of potassium, well mixed and sifted.

In the form of cigarettes, datura tatula, strammonium, tobacco, and many other substances are employed; I have

chiefly used the second. In one case I used arsenical cigarettes, but without noticeable benefit. Nitrite of amyl, 4 or 5 minims dropped upon a handkerchief and inhaled, has been found a serviceable remedy, but its action will be best considered when we come to nitro-glycerine. Germain Sée has obtained very good results from inhalations of iodide of ethyl. It can be given very conveniently in the glass capsules, each containing 5 minims.

Of the use of ether and chloroform I cannot approve; they are too dangerous to be entrusted to the patient or his friends, and their seductive effect is so great that the patient who uses either of them runs the risk of being constantly under their influence.

Among the drugs used subcutaneously morphia is surpassed by none; even Dr. Berkart,* who looks askance at most of the therapeutic measures employed in asthma, has a good word for morphia, and says, "In no way can the bronchial tenesmus be so rapidly and so safely subdued as by the subcutaneous injection of morphia," and he goes on to say, "There is, so far as my experience goes, no contraindication;" but he very rightly adds, "Nor does anything deserve so strongly to be condemned as the habit of entrusting the patient or his nurse with the syringe and the solution." In this statement I heartily concur. The use of morphia subcutaneously is much limited, as it is not often that the medical man himself has the opportunity of personally treating his patient during the attack. If morphia is employed it is best given in combination with atropine—$\frac{1}{4}$ grain of morphia with $\frac{1}{150}$ of atropine. Salicylate of cocaine has been used with excellent results by Professor Mosler,† of Greifswald.

Among drugs given by the mouth I would assign the first place to nitro-glycerine. My attention was first directed to the beneficial action of the nitrites and the nitrite-acting substance, nitro-glycerine, in the dyspnœa of asthma and bronchitis by a paper of Professor Fraser in the *Lancet* of July 9th, 1887. Since this time I have constantly used them, but especially nitro-glycerine. The latter is the most convenient for use, as it is perfectly stable, can be readily taken by the mouth, unlike

* 2nd edition, p. 181.

† British Medical Journal, vol. ii. p. 117.

the nitrite of amyl, which is so volatile that all persons in the neighbourhood of the patient are liable to be affected by the vapour given off. I usually order a minim of the 1 per cent. alcoholic solution of nitro-glycerine in half an ounce of dilute chloroform water to be taken immediately the paroxysm comes on, and to be repeated in half an hour or an hour according to the urgency of the symptoms. It is advisable to begin with a small dose on account of the severe effects produced in some people by nitro-glycerine. The alcoholic solution acts more quickly than the tabloids.

The following are some of the cases in which I have given nitro-glycerine for the relief of dyspnœa in asthma and bronchitis.

Mary Ann C. Aged 45. Suffering from bronchitis and emphysema with asthmatic paroxysms. This patient derived great benefit from $\frac{1}{200}$ grain of nitro-glycerine, relief usually coming on within half an hour; she says that the drug loosens the cough and diminishes the wheezing.

Jane E. Aged 39. Suffering from bronchitis and emphysema with asthmatic paroxysms. Ordered to take ammonia, senega, and nux vomica three times daily, and cod liver oil twice daily. On November 17th, 1887, $\frac{1}{200}$ grain of nitro-glycerine was ordered to be taken every hour for the asthmatic paroxysms, and she derived great benefit from it.

On October 18th, 1888, she stated that she experienced "wonderful relief" from the nitro-glycerine.

November 8th. Much worse, as she has been unable to come and get the nitro-glycerine.

November 15th. Has taken the nitro-glycerine again with relief in about five or six minutes.

F. C. Female aged 44. Came under treatment in June, 1887, for attacks of dyspnœa occurring at night; these came on about 2 A.M. at intervals of two or three months; the last two attacks occurred at intervals of a month, and the last attack persisted for about forty-eight hours. During the attack her breathing is short and gasping, and she has a slight cough but without expectoration; as soon as expectoration comes on she gets great relief. Examination of the chest revealed nothing abnormal.

She was ordered 5 grains of iodide of potassium with 3 grains

of carbonate of ammonium three times a day, and 15 minims of the ethereal tincture of lobelia in an ether mixture every three hours during the attack. No benefit resulted from this treatment, and on October 13th, 1887, as the attacks were occurring nightly she was ordered to take $\frac{1}{200}$ grain, afterwards increased to $\frac{1}{100}$, of nitro-glycerine in alcoholic solution every hour so long as the dyspnœa lasts, and to continue the iodide of potassium mixture.

On October 20th she reported that only once had she occasion to take a second dose, relief coming within the hour. The last time this patient was seen was in November, 1889; the attacks had been much less frequent. At one visit she said that there had been an interval of six months of freedom, and that the $\frac{1}{100}$ grain of nitro-glycerine still gave almost immediate relief.

Professor Fraser's observation that some of the dyspnœa of bronchitis is due to spasm of the tubes is a valuable addition to our knowledge, and I have found great advantage in the administration of small doses of nitro-glycerine ($\frac{1}{200}$ grain) in these cases. Even smaller doses than this act usefully, and patients say that the medicine loosens their cough, and that they breathe easier after it.

Citrate of caffeine is another drug of which I have had very favourable experience in asthma. I usually give it in 3 grain doses in combination with 20 grains of bromide of potassium—the addition of the latter prevents the insomnia which caffeine, when given alone, often produces. In one patient, in particular, in whom the attacks had been preceded by sneezing and profuse defluxion from the nose, the mucous membrane being much hypertrophied, the effect of the caffeine was so marked that I gave up the intention I had formed in my own mind of using the galvano-cautery. The patient suffered from almost nightly attacks, and when seen three years after the caffeine had been ordered she stated that she had hardly any trouble with the asthma, and that the medicine relieved her greatly.

Dr. Samuel West* records the case of a lady, aged 63, suffering from chronic bronchitis and asthma. One-sixth of a grain of pilocarpin by the mouth gave almost immediate relief. In a few minutes the patient was in a state of comparative comfort and rapidly improved. In two or three subsequent attacks a similar effect was produced by the same dose.

* St. Bartholomew's Hospital Report, vol. xx. p. 126.

Though I have come across several patients who have taken chloral with advantage I have not myself ordered this drug for the relief of the asthmatic paroxysm.

Favourite domestic remedies are hot black coffee and a glass of hot grog.

Having mentioned drugs which I have found useful, I will now draw attention to one only to condemn it. This is lobelia. I have had two unfavourable experiences with it, and in one unfortunately the result was fatal. The first case came under my notice while I was House Physician at St. Bartholomew's. Ten minims of the ethereal tincture of lobelia were ordered to be taken every four hours by a patient suffering from chronic bronchitis with asthmatic attacks. After the second or third dose she became violently sick, broke out in a cold sweat, and became almost pulseless. The application of hot bottles and the administration of diffusible stimulants sufficed to restore her strength, though at the time I feared a fatal result. In the second case, a lady of 48, suffering from a very sharp attack of asthma, took 10 minims of the ethereal solution of lobelia, repeated it in an hour, and then every four hours; she died after taking a few doses, and I am convinced that her death was partly due to the lobelia. Since that time I have not prescribed lobelia in cases of asthma. To get any decided effect from it, the drug must be pushed, and there is great risk of dangerous depression of the heart's action. Dr. Nunès,* of Rio, however, has made an important observation on the action of lobelia. He attributes the emetic action of the tincture to the form of preparation. The solvent employed separates from the plant an emetic principle different from lobeline, the principle beneficial in asthma. He states that lobeline does not produce in medicinal doses either nausea or vomiting, and that its action is rapid and evanescent. It may be given by the mouth, or subcutaneously, in doses of from $\frac{3}{4}$ grain to about 6 grains.† If the good results which Dr. Nunès has obtained be verified we shall have gained a valuable remedy in the treatment of asthma. The action of the non-depressing emetics is not to be condemned, as they are sometimes of great service in cases in which the asthmatic paroxysm has supervened

* British Medical Journal, 1889, vol. ii. p. 1051.

† Bartholomew gives the dose of lobeline at $\frac{1}{100}$ to $\frac{1}{20}$ of a grain, but this can hardly be the same substance as that used by Nunès.

upon a full meal. I remember, in particular, one case in which the patient said he had derived more benefit from mustard taken as an emetic at the onset of the paroxysm than from all other kinds of treatment.

FIBROID TUMOURS OF THE UTERUS

TREATED BY

ELECTROLYSIS.

FURTHER CASES.

By Dr. POTTER.

In the last volume of the Westminster Hospital Reports (p. 129) I placed on record three cases treated by electrolysis; I now add four more occurring during the present year.

Case 1.—E. M., æt. 46, single, admitted October 12th, 1889.

Symptoms complained of on admission: faintness, sickness, and dragging pain in the abdomen, with increase of size and excessive loss at the catamenial periods. These symptoms were first noticed twelve months ago.

Family history.—Mother died at 65 of Bright's disease, having also suffered with an abdominal tumour.

Present state.—October 15th. There is found on examination a tumour in the abdomen which reaches in the middle line to the umbilicus; higher on the left, lower on the right. Its surface is fairly smooth and regular.

Circumference.—Most prominent part, 30 inches; at umbilicus, 27 inches; right anterior spine of ilium to umbilicus, 6 inches; left anterior spine of ilium to umbilicus, 6½ inches.

Per vaginam.—Cervix high up; os small, points backwards; cervix small, considerable swelling in front, continuous with that felt in abdomen. Sound passes at first backward and then turns in a forward direction, altogether measuring 3½ inches.

October 16th.—Battery, 170—140 milliamperes.

18th.—Battery, 100—170 milliamperes. Bad night after.

25th.—Catamenia appeared (18 diapers used).

November 12th.—Battery for the 5th time. Circumference at most prominent part, 29½ inches; transverse diameter of tumour, roughly, 6½ inches.

17th—23rd.—Catamenia very profuse.

December 10th.—Battery, 10th time; sound passes 3½ inches.

10th—16th.—Catamenia about the same as last time.

31st.—Battery, 16th time.

January 2nd—7th.—Catamenia more than usual (25 diapers). At this time (7th) the tumour extends in the middle line and to the left of the same level just below the umbilicus. On the right it is two fingers' breadth below, and extends 3 inches from the middle line; to the left it reaches quite into the flank, almost to the spine. Character of tumour not altered.

24th.—Battery, 21st time.

27th.—Measurement at umbilicus, 27 inches; most prominent part, 29½; iliac spines to umbilicus, 6½ inches each.

29th—February 4th.—Catamenia.

January 31st—February 7th.—Eruption of erythema nodosum on both legs, with rise of temperature to 101°. At this time it was noticed that a portion of the tumour at right lower edge, about 2 by 2 inches, was distinctly softened.

February 7th—27th.—Nodes subsided, followed by erytheme exudativum, with marked œdema of both legs. During these attacks the use of the battery has been discontinued.

11th.—The tumour is one finger's breadth below the umbilicus in the middle line, on a level with it to the left, and on the right 3 inches below. At umbilicus, 27¼ inches; most prominent part, 29¼ inches.

21st.—Tumour has same position, but more marked feeling of softening at its right edge.

27th—March 3rd.—Catamenia much less in quantity, and only lasting four days.

March 4th.—At umbilicus tumour two fingers' breadth below, one on left, and same distance on right. At umbilicus circumference 26 inches; most prominent part, 27½ inches; rough transverse, 5 inches.

7th.—Battery, 23rd time.

25th.—Tumour midway between pubes and umbilicus, more evident on left than right side. Transverse measurement, 4 inches.

April 3rd.—Battery, 30th time. Hæmorrhage, ? catamenia, 4th—8th.

8th.—Tumour one finger's breadth below umbilicus, and on a level with it on the left side. Transverse diameter, roughly, 7¼ inches; circumference at umbilicus, 27 inches; at most prominent part, 28½ inches.

9th.—Patient returns home.

This patient, previous to her admission into the Hospital, had been under observation and treatment since March, 1889. She had then felt the enlargement about three months, noticing that her dresses were feeling tight. She did not then notice it more on one side than the other. She thinks the cause to be due to long nursing and overlifting. Catamenia were quite regular up to this time; since then the intervals have been less, and the periods longer. Pain is not a marked symptom unless she lies on her left side, then after a feeling of sickness it becomes of a dragging character.

At this time (March) abdominal examination reveals a tumour hard in character and extending to two fingers' breadth below the umbilicus, more to the left side than the right. Surface rather irregular. Vaginal examination difficult from presence of hymen; os points to the left and is reached with some difficulty; tumour is felt more to the right, impulse with abdominal tumour distinct.

She was now treated with ergot, also bromide of potassium at bedtime.

June 8th.—She has felt better, been regular, and not lost so much at the periods. She thinks she gets larger, and the pain continues. The abdominal and vaginal examination is much as before. Is to take the bromide twice a day.

October 5th.—Catamenia have appeared five times in the last four months, very considerable in quantity and with more pain. Faintness and retching at times. Bowels only act now with medicine. The last period finished five days ago.

Abdominal examination.—The tumour extends now nearly to umbilicus, more to left than right side. *Per vaginam.*—Sound passes with much difficulty 3½ inches in a forward direction.

Patient left the Hospital on April 9th.

On May 29th she is seen, and the following note made:—The tumour extends to two fingers' breadth below umbilicus, is

narrow across, but prominent. *Per vaginam.* The uterus does not press down so much, though the cervix is still squeezed to left. Last period occurred one week ago after an interval of six weeks; lost a good deal, and has had much pain at the time.

CASE 2.—L. L., æt. 40, married. Admitted into Hospital December 5th, 1889.

Patient complains of repeated hæmorrhages at irregular intervals for the last three years. Worse the last nine weeks. The hæmorrhage is always very profuse, and followed by diarrhœa.

Abdominal examination.—The uterus is felt 2 inches above the pubes.

Vaginal examination.—Cervix high up, rather hard, uterus heavy but movable. The sound passes 3½ inches in a forward direction.

The treatment consisted of rest, hot douches (110°—115°), and the administration of ergot.

January 6th.—Uterus heavy; os admits the tip of the finger as far as the second joint, walls of cavity smooth. From anterior wall projects a softish mass, irregular in shape and the lower portion of it free.

February 7th.—It was determined to explore the cavity more thoroughly, and this was done after dilatation with Hegar's dilators, under ether; a tumour was felt about the size of an orange occupying for the most part the posterior wall of the uterus; it was adherent except to the extent of ¼ inch in front, near the os, where the finger passes between it and the wall of the uterus.

21st.—Battery used for the first time, 150 milliamperes, for ten minutes. A good deal of blood came away after the application.

March 10th.—Battery has now been used 5 times. Period commenced to-day, continued slightly until 14th.

28th.—Battery used for the 10th time; the number of cells required to produce an average of 150 milliamperes has varied from 28 to 42.

April 1st.—Hæmorrhage profuse with large clots. Bimanually the uterus seems smaller.

May 2nd.—Battery has now been used 14 times. Sound passes in forward direction 2½ inches. Patient goes to-morrow to a convalescent home.

CASE 3.—H. G., æt. 44, married; no children; 1 miscarriage. Admitted into hospital January 13th, 1890.

Patient complains of constant hæmorrhage, pain in back and hypogastrium.

Abdominal examination.—A tumour is found which extends in the middle line up to the umbilicus, slightly above it on the left, and is about the umbilical level to right of the middle line. Tumour otherwise fairly symmetrical, extending about an equal distance on both sides. Flanks fairly resonant, centre dull. Dulness wooden two fingers' breadth below umbilicus.

Vaginal examination.—Cervix in fair position; os small; uterus somewhat fixed, enlarged, and hard all round, especially in front and to the right. Sound passes in backward direction and to right for 2½ inches, then turns forward and passes a further 3 inches, making 5½ inches in all. Cavity not enlarged, a good deal of bleeding going on.

January 16th.—Hæmorrhage continues profuse.

January 17th.—Sound passed 5½ inches. Battery used for ten minutes, 28 cells=150 milliamperes.

January 22nd.—Circumference of abdomen at umbilicus, 31 inches. Two inches below, 33 inches; and right and left iliac spines to umbilicus equal 7 inches.

January 27th.—Complains of pain and tenderness over abdomen, increase of hæmorrhage and sickness. So battery was not used to-day.

February 4th.—Battery to-day for the 5th time.

February 6th—11th.—Catamenia flowing. Measurements at umbilicus, 30¾ inches; most prominent part, 32 inches.

February 28th.—Battery for the 10th time. Cervix thickened and more irregular.

March 18th.—Battery to-day for the 15th time. The tumour is much smaller, being half-way between the pubes and umbilicus. Measurements at umbilicus, 30½ inches; most prominent part, 32¼ inches in middle line; the tumour is four fingers' breadth below umbilicus. Sound passes 5½ inches.

April 1st.—No period during March. Tumour is three fingers' breadth below umbilicus in middle line, and is more diffused. Measures, roughly, 5 inches transversely.

April 8th.—Tumour within two fingers' breadth of umbilicus, not clearly made out. Transverse diameter, roughly, 6 inches;

circumference at umbilicus, 30½ inches; most prominent part, 32½ inches.

April 12th.—Patient complains of sickness and pain in the back.

April 25th.—Battery, 22nd time. Tumour not clearly made out, 3 inches from umbilicus. Measurements—transverse, 4½ inches; at umbilicus, 31 inches; most prominent part, 32½ inches. Sound still passes 5⅛ inches.

April 29th—May 4th.—Catamenia.

May 13th.—Battery for the 26th time. A little bleeding. Tumour extends to within 1 inch of umbilicus. Measurements at umbilicus, 31 inches; most prominent part, 32 inches; transverse, rough, 6 inches. Good deal of blood and clots after application to-day. Patient goes home to-morrow.

Case 4.—E. C., æt. 35, married; 1 child; 3 miscarriages at 2, 6, and 7 months. Admitted March 10th, 1890.

Patient complains of hæmorrhage on and off the last four years, following the birth of her child. It has been getting worse the last six months.

Abdominal examination.—No tumour to be felt.

Vaginal examination.—Uterus high up, cervix rather hard, os irregular. Body of uterus feels heavy, but is fairly movable. Bimanually fundus is felt midway between pubes and umbilicus. Sound passes easily 3½ inches in a forward direction.

March 14th.—After dilating cervix, cavity of the uterus examined with the finger; is found to be quite smooth. Bimanually a small hard tumour, the size of a Tangerine orange, is felt in the wall of the uterus to the right and front.

March 21st.—Went home on account of domestic affairs and returned on

April 7th.—The day after battery is used for the first time, sound passed 3¼ inches; 30 cells=150 milliamperes, were used for ten minutes.

April 25th.—Battery for the 6th time. A good deal of brownish discharge, and one or two very small clots.

May 8th.—Patient went home of her own wish, the battery having been used eight times.

June 3rd—Patient reported herself; states that the catamenia came on May 25th—31st. Not profuse and without pain.

It is difficult to comment on these cases with any degree

of certainty. It will be noted that in three of the four the age was 40 years and upwards—40, 44, 46. In such cases, of course, the possible nearness of the menopause has to be taken into consideration. In all the cases, as in the three previously recorded, the procedure was the same. The introduction of a platinum sound into the uterus as far as possible, twice a week, and the strength of the current (positive pole) being an average of 150 milliamperes. In the first and third cases there seemed at one time evidence of decided diminution in the size of the tumours, but this was only transient; the amount of bleeding, too, has varied very considerably. In the other two cases, which were instances of small interstitial fibroids, the bleeding was certainly diminished. On the whole I am inclined to think that the benefit is mainly due to galvano-caustic action, and that probably a similar result might be obtained with treatment less expensive, troublesome to manage, and cumbrous.

For the notes of these cases I am indebted principally to Mr. Burt and Mr. Dawson, and for help in the use of the battery to Dr. Wills.

COMMENTS ON SOME INTESTINAL DISORDERS OF CHILDHOOD.*

By Dr. DONKIN.

Enteritis.

We find in the post-mortem examination of children who have died from various diseases, with or without diarrhœa, a marked catarrh of the intestines, and sometimes a severe enteritis with extravasations or fibrinous exudation. I have once seen hæmorrhagic enteritis of the ileum and colon in a case of febrile wasting where tubercle was suspected, but nowhere found. Henoch reports two cases of marked gastro-enteritis without intestinal symptoms in connection with chronic nephritis, in one of which the lower part of the ileum was covered in places by a coherent fibrinous membrane; and Goodhart quotes one of severe "diphtheritic" inflammation of the colon and rectum where there was towards the end some watery diarrhœa with slight melæna. *Tubercular ulceration* is often found post-mortem in cases unmarked in life by diarrhœa or other intestinal symptoms.

True *dysenteric disease* of the intestine is very rare in English children who have not lived in countries where the disease is endemic. I have seen some marked cases of young children who began to suffer after returning from the East Indies where they had all recently had malarious fever. A remarkable series of cases of what was symptomatically acute dysentery was admitted into the East London Hospital for Children in the summer of this year. The four patients were of one family, varying in age from one and a half to eight years, and the father was affected as well. All the cases occurred between the 17th August and the 13th September. The father and the youngest child died.

* This paper is the substance of some clinical lectures.

In all the disease began suddenly with much abdominal pain, profuse diarrhœa, and tenesmus with the passage of blood and slime, and, in the later stage, of shreddy material or obvious sloughs, with no fæces. In most there was vomiting at the outset, in the elder ones shivering, and in all rapid wasting. Convalescence is very slow in the three cases still under treatment. Inquiry revealed no dietetic cause for the disease, nor other source of poisoning, with the important exception of the discovery by the father soon after his attack of a much decomposed rat in the cistern, the water from which had previously "tasted very bad." The mother, who did not drink water, and three other children who did, escaped the disease. In the fatal case marked ulceration and sloughing patches were found along the whole length of the large intestine.

There is nothing in dysentery special to childhood, as regards either diagnosis or treatment. I believe bismuth and opium to be the most valuable drugs in this disease, the bismuth (subnitrate) being given in large doses and in water only; and starch and opium or other astringent enemata are occasionally invaluable. Much attention must be paid to diet and general hygiene for a long time after convalescence is established.

Gastro-Intestinal Hæmorrhage.

Neither hæmatemesis nor melæna to any extent are very common occurrences in infancy or childhood. These symptoms are, however, sometimes important, and may be due to various causes. Epistaxis may, if profuse, occasion both symptoms. Oral and faucial ulceration sometimes give rise to bleeding which may colour the vomit from accidentally concurrent gastric disturbance, as pointed out by Eustace Smith. Both hæmatemesis and melæna from disease of the stomach itself are very rare, but either or both may occur in infants in their first week or a little later. Most of such cases die with symptoms of collapse, and it is said that those which recover do not show any after tendency to hæmorrhage. Various anatomical conditions have been put forward to account for this affection. Small ulcerations, both single and multiple, are not infrequently found in the stomachs of infants, but in most of these cases there has been no hæmorrhage. Dr. Goodhart in 1881 reported one case in an infant two days old where a small gastric ulcer was found to be

the cause of fatal hæmorrhage from mouth and anus; and Henoch gives an instance of hæmatemesis and melæna in a child of five years old where there was ulceration of the cardiac end of the œsophagus. Capillary hæmorrhage alone has been seen or inferred in the majority of cases, and is usually attributed to increase of venous pressure from any cause interfering with respiration.

Sometimes infants vomit or pass per anum a little blood which has been swallowed from sucking sore nipples. The hæmorrhagic diathesis or purpura may cause among other bleedings, both hæmatemesis and melæna in a marked degree in children beyond infancy. The diagnosis is generally easy owing to the concurrence of cutaneous eruption or of bleeding from the nose or gums or the buccal mucosa. In the malignant forms of some of the exanthemata gastro-intestinal hæmorrhage is occasionally observed, especially in small-pox.

The more common causes, however, of these symptoms are local, as in ulceration or severe catarrh of the intestines, tubercular disease, enteric fever, or more rarely, dysentery. Ulceration of the stomach, so common in youth and middle age, is but very rarely seen in children, and has no claim to further regard here. Melæna is a prominent symptom of great diagnostic import in intussusception, results often from a polypus in the rectum especially during or after defæcation, and may accompany prolapse of the bowel.

The blood which comes from local lesions may be bright or dark according to the seat and nature of the lesion, and mixed or unmixed with fæces. In intussusception and rectal polypus the blood is usually red; in ulceration higher up it is generally dark. In every case of gastric or intestinal hæmorrhage a careful search for a local source must be made before making any definite diagnosis from inference alone. The rectum should always be examined. I once saw post-mortem in a case of enteric fever, where death occurred a day or two after profuse melæna, two deep ulcers a little above the sphincter, which were the undoubted source of the blood. Ulceration elsewhere was slight and superficial. The hæmorrhage here might possibly have been checked by local treatment had the rectum been examined. In the severe forms where no local cause can be detected or reached, such as in the melæna of the new-born, an ice-bag

should be applied to the abdomen and small quantities of iced milk given by the mouth. The child should at the same time be stimulated by alcohol and kept warm. It is useless in severe cases to waste time over the trial of medicinal styptics given by the mouth, their action being at the best highly uncertain. The subcutaneous injection of ergotin, from ¼ to ½ grain, should be always used. The same treatment is applicable to all cases with similar symptoms whatever their cause may be. Whenever examination detects local disorder, local treatment is necessary, such as the removal of a polypus, or the injection of some astringent remedy, as starch and opium or a solution of nitrate of silver, into a rectum which is the subject of ulceration or severe catarrh.

Prolapse of Rectum.

This is a very common affection in infancy and very early childhood, and is generally characterised by the invagination of the middle in the lower part of the rectum, and its protrusion through the anal ring in the form of a shiny red swelling which readily bleeds. A small prolapse returns by itself after defæcation, while larger ones require replacement. Although some cases are doubtlessly referable to straining with constipation, or to the lax and catarrhal condition of the middle part of the rectum in connection with severe diarrhœa, I am convinced both from examination and inquiry in numerous cases of out-patients that this affection very frequently occurs with no intestinal disorder and is probably due to a feeble sphincter. It must be remembered, too, that the rectum is straighter in infancy than later on, and that the sigmoid flexure is in the middle line or sometimes a little to the right. Straining in urination may excite prolapse in time, and it is generally recognised that stone in the bladder should be sought for as a possible determining cause of prolapse.

Some cases get well with only a few replacements, others may be very obstinate indeed, but most recover in the long run. The treatment is to return the tumour by firm pressure with one or two fingers on its central part, and then to apply a thick pad to the anus, bandaging or strapping the buttocks tightly together. Henoch gives a qualified recommendation to the subcutaneous injection of ergotin in the neighbourhood of the anus,

stating that it never does local harm, often seems to do good, but often fails. Nitrate of silver in strong solution may be applied with frequent success to the surface of the tumour. In some cases surgical treatment directed to the contraction of the sphincter ani is found necessary. Concurrent constipation or diarrhœa should be always appropriately treated.

Constipation.

By this term I mean insufficient evacuation of the bowels as evidenced by any one or more of the following symptoms: pain or local discomfort during defæcation, prevailing abdominal distension or discomfort, with small stools (not always either hard or very infrequent), or certain symptoms of ill-health concurring with markedly deficient action of the bowels. Merely infrequent bowel action is physiological with many children, and when unattended by any local or general signs of disorder is neither to be dreaded nor treated. Constipation, medically speaking, connotes some other morbid symptom or state, and may be caused by deficient expulsive action of the intestines or by some obstruction in their course. I shall include here, however, only those cases which are unconnected with deformity or mechanical lesion.

Some infants have deficient intestinal action from birth, even when breast-fed; the stools are lumpy and hard, passed with straining, often very pale or almost colourless, like the stools of jaundice, and sometimes slightly streaked with blood from small erosions within the anus probably caused by the hardened fæces. Constipation in infancy, a period when the bowels are, as a rule, frequently evacuated, should always be noted. Its cause is often not to be detected, for though there may be sometimes signs of catarrhal derangement of the intestines, or of deficient action of the liver, or possibly of the pancreas after such time as the pancreatic secretion is usually well established, in very many cases the only symptom is the difficult or painful passage of hard dry fæces, and the dryness of the fæces is by no means always explicable by drain of fluid from skin or kidneys. Unquestionably a change in the diet may indicate the cause while working the cure of constipation, and such change should always be tried. Often the tendency remains till the child takes active exertion and a mixed diet, and is probably due to generally deficient nerve force causing intestinal atony. The frequency of constipation as

a symptom of brain disease, as in tubercular meningitis, must be remembered in this context, and its very general concurrence with melancholia, hypochrondriasis, and other evidences of a disordered nervous system in adult life. When local and dietetic causes can be excluded by careful examination and reflection on the case, we must trust to time and general tonic measures, with such stimulation by food as may be consistent with the child's age, to help on the cure. Frequent and prolonged kneading of the abdomen with the oiled hand is often very useful, and good is sometimes gained by occasional small suppositories of soap or injections of cold water, or of half a drachm of glycerine. In infants there is little advantage in the use of medicines, which should be always avoided if possible. Sometimes, however, a purge will be found necessary when distension or discomfort is great. The sluggishness of the bowel may, perhaps, be lessened by the systematic administration of liquor strychnia or tincture of nux vomica, or by tincture of belladonna; there is, at least, a considerable amount of authority in favour of the treatment, but I have seen little or no result either in children or adults with habitual constipation from the persevering use in many cases of such remedies, although in two instances (in an adult and a child respectively) of marked constipation with flatulent distension I have observed a rapid diminution of the abdominal swelling with expulsion of gas follow soon after the administration of one or two full doses of strychnia. Occasionally in infants constipation is due entirely to irritation at the anus, caused by painful fissures, and inducing contraction of the sphincter. Laxatives and local treatment by nitrate of silver or some other astringent, or the application of cocaine, or, failing these remedies, surgical treatment of the fissure must then be resorted to at once.

Apart from these instances of infantile constipation which frequently lead to no further symptoms and do not necessarily interfere with health, it may be said that constipation at all ages owns various causes, and each case must be studied and treated by itself. The chief causes, outside mere idiosyncrasy, are want of exercise, too unstimulating or easily digestible or *monotonous* diet, and overfeeding with neglect of regular times for defæcation. A diet largely composed of milk greatly favours constipation, as also does prolonged rest in bed. This is seen

markedly in most cases of patients confined to bed, in convalescence from acute diseases, and especially from enteric fever. Sometimes constipation of considerable persistence is observed after severe attacks of diarrhœa, or the frequent unnecessary use of purgative drugs. In these latter cases time and a mixed diet are at once the most rational and successful medicines.

Habitual or marked constipation at any period of infancy or childhood may be accompanied by local symptoms of more or less severity, or general ill-health, or a combination of these conditions. When the constipation from whatever cause is marked or lasting there may be distension of the abdomen with tenderness, giving rise to a suspicion of peritonitis. Great loading of the lower bowel with fæces may be attended by the daily or more frequent passage of small liquid stools, sometimes amounting to what may be called diarrhœa. In small degree these cases are not rare. An extreme example, ending fatally, I once saw in a boy about three years old who had been medically treated without examination for some months for diarrhœa of over a year's standing. He was admitted into hospital in a state of collapse with much pain and enormous distension of the abdomen, and died suddenly a few hours afterwards. The bowel was found post-mortem to be greatly stretched and full of a rock-like mass of hard grey crystalline fæces which nearly filled an ordinary bucket. In the mass there was a small sinuous channel through which the liquid evacuations, fatally diagnosed and treated as "diarrhœa," had been constantly trickling. The diaphragm was pushed far up into the thorax, and the boy had died from cardiac paralysis. Eustace Smith relates a very similar case. The lesson is here taught to make a thorough physical examination of all cases of reputed diarrhœa, as well as of constipation, instead of trusting to hearsay evidence before instituting a course of treatment. Constipation *per se* should never be fatal either directly or indirectly, nor even give rise to serious symptoms. Complete occlusion of the intestine and typhlitis leading to perityphlitis or general peritonitis, are said by various authorities to be the occasional or frequent results of neglected constipation. These events are, nevertheless, exceedingly rare, or altogether apocryphal. Constipation preceding typhlitis is not, as often taught, the rule, but the exception, and this occasional connection is merely accidental.

As a result, however, of typhlitis beginning generally in the vermiform appendix constipation is often marked, and sometimes favours the mistaken diagnosis, and leads to the surgical treatment, of occluded bowel.

The symptoms, other than local, which may accompany habitual constipation are numerous and often indefinite, and require much care before being referred to any one cause. Many of the ailments attributed to constipation are doubtless part of the original malady out of which the constipation arises, as is exemplified in some neurotic cases, and in *rapidly developed* anæmia. Decided rise of temperature is a frequent accompaniment of a loaded bowel. Not only in enteric fever, both during its course and in convalescence, but also in apparently simple cases, there is abundant clinical evidence of raised temperature resulting from constipation. From among numerous examples of this in my note-books I give the following extraordinary instances of two young children who were admitted at different times into Hospital with great abdominal distension and pain, and temperatures of 107° and 108° respectively, in both of which all these symptoms rapidly and permanently disappeared after the evacuative action of simple enemas. I am inclined to refer such cases as this, with other instances I have seen of abdominal pain at all ages accompanied by temporary high temperatures, to a nervous origin through impressions made on the abdominal sympathetic, by reason of the suddenness of the rise of temperature and the equal suddenness of its fall, with coincident evacuation of the bowel. In more chronic cases of constipation some pyrexia may be due to the retention of excrementitious matter, or "auto-infection," but I feel sure that this possible event may be made too much of by the modern physiologist and physician. For the rest, as in adults, so in children, habitual constipation is often accompanied, be it causally or coincidentally, by a sallow or pale complexion, by languor, slow circulation, loss of appetite, foul breath, troubled sleep or headaches; and there are some cases where such symptoms disappear if the constipation be cured by diet or drugs. Recurrent sick-headache, or migraine—a very common disorder of childhood and almost always the outcome of a special or general hereditary neurosis—is not seldom attended by, and in my opinion most erroneously referred, both by the

profession and the public, to constipation or indigestion, the attempted relief of which in no way lessens the frequency or duration of the other symptoms.

The general *treatment* of constipation, after a careful inquiry into its most probable causation, is both hygienic and remedial. In all cases the abdomen must be examined, when scybala may often be felt; and the rectum explored, and if loaded, evacuated by an enema or other means. In recent acute cases, accompanied by abdominal pain or other urgent symptoms, rest, opium, and expectance are the next steps, as will be further mentioned under the head of Obstruction. In the absence of vomiting or other symptoms of obstruction, and especially of intussusception, even when there is pyrexia, distension of the abdomen with a history of constipation sometimes indicates, as I have above shown, evacuation by enema. Even if there be mechanical obstruction one determined effort with this object will probably be harmless. No definite directions, however, as to the due diagnosis of such cases can be given.

In chronic constipation the diet must be first attended to. I have already indicated the general treatment of infantile constipation which rarely requires aperients. But if there be reason to believe from the concurrence of gastric disorder or the appearance of the stools that a catarrhal condition is present, the infant's diet should be regulated accordingly, and milk and farina should for a while be given sparingly or not at all. Small doses of sodium bicarbonate and rhubarb will then often be found useful for a few days. If the supposed catarrhal condition of the intestine be due to chill either as a symptom *per se*, or a part of more widespread evidence of cold, warm clothing to the body, and especially the abdomen, is certainly indicated. At all events, however doubtful the exact diagnosis may be, chronic constipation is so often attended by sluggish circulation that due attention should always be given to this point. Whether or not deficient fluid be the cause of constipation a free supply of pure water may always be tried in cases where the stools are dry and hard; it is at least a good stimulant to a torpid intestine. In older children a daily regular evacuation after breakfast, or an attempt at it, should be insisted on both as a preventive and remedial measure; many troublesome cases of constipation are initiated by neglect of this custom otherwise so important in

civilised society. A mixed diet with plenty of vegetables and fruit and whole-meal bread is necessary, and exercise with gymnastics suited to the age is to be enforced. All hygienic measures should be taken before resorting to drugs, which are, however, in some cases required not only to remove the abdominal discomfort which sometimes results from a loaded bowel, but also to prevent the possible ulterior symptoms of ill-health which have been mentioned. Merely purgative drugs, however necessary they sometimes may be, are but temporarily stimulant; they should be used in the smallest doses which have the desired effect, and as seldom as possible. I believe that senna and sulphur in the form of confections, or aloes, are as good remedies as any, and cascara is often very effective. I have already said that in spite of very many cases of habitual constipation in children being in my opinion primarily referable to nervous deficiency and not to local disorder or disease of the alimentary canal, I have usually been disappointed with the much-praised action of strychnia and belladonna so widely used in this affection. It is of the highest importance to attend to all concomitant symptoms in cases of chronic constipation. I have had far greater success from the prescription of exercise, sunlight, fresh air, arsenic, cod-liver oil, and iron, and the proscription of prolonged rest, either in bed or out of it, than from any systematic course of special drugs. There is in fact but little peculiar to childhood in constipation which has much the same clinical aspects at all ages. There are few affections where the therapeutist's temptation is so great to achieve an easy and superficial success by symptomatic treatment while neglecting the causal conditions of the disorder.

Intussusception and Intestinal Obstruction.

The subject of mechanical obstruction of the bowels in young children, apart from the matter of fæcal accumulation already dealt with, practically resolves itself into that of intussusception. Occasionally we meet with other occlusions, such as partial or complete congenital stricture, which may sometimes be due to fœtal peritonitis, hernia, strangulation from peritoneal bands, and obstruction from malignant or other tumours; but these, with the exception of complete congenital atresia, present nothing special to childhood. It must never be forgotten that symptoms

of acute peritonitis, from whatever cause arising, very frequently almost exactly simulate intestinal obstruction.

Intussusception is most common in infancy and early childhood, and at this period generally consists in the invagination to a greater or less extent of the lower part of the ileum and ileo-cæcal valve within the colon, the cases of engagement of the small intestine alone being mostly confined to later childhood and adult life. The comparative frequency of the involvement of the cæcum in infancy has been referred to its looser connections in the iliac fossa at this age. The anatomy of intussusception is amply described in the general text-books. I would but mention here that small and multiple intussusceptions, easily reducible, are frequently found post-mortem, and doubtless take place during the act of dying. Those which cause symptoms are larger, at least two or three inches in length, and may involve several feet of gut, and be felt in the rectum, or even protrude from the anus. No exciting cause for the disordered peristalsis which must precede this affection can be demonstrated in most cases. Eustace Smith quotes cases occurring soon after a fall, and I have seen two instances of this connection which may or may not be causal.

Intussusception leads to partial or complete obstruction of the canal and inflammatory strangulation of the two contained layers of bowel. Sometimes ulceration takes place, and perforation through the outer layer into the peritoneal cavity, or there may be general peritonitis without perforation. The invaginated part may be so gripped as to quickly become gangrenous, and may be discharged through the anus. In some cases with firm adhesions recovery takes place, the external layer of bowel forming a tube continuous with the unaffected part above the lesion.

Clinically cases of intussusception may be divided into two classes—those where the diagnosis is almost certain, and those where it is only probable. The clearest cases are those where the child shows sudden evidence of abdominal pain, screaming, and drawing up the legs; the pain comes on paroxysmally, vomiting follows, which is repeated, and there is straining with the passage of fæces if the bowel below the obstruction be not already empty, or with the passage of mucus or blood. There is generally no marked abdominal tenderness at the outset, and

deep pressure can be made without finding any tumour. Later on a tumour, tender on pressure, may sometimes be felt externally, if the abdomen be not distended, and in the course of time a soft elastic swelling may be detected by the finger in the rectum. After one or perhaps more evacuations at the outset constipation nearly always sets in. The child is very restless, and may not sleep at all. If the symptoms do not soon remit with or without treatment they rapidly increase. Coils of dilated intestine are often seen through the walls, collapse takes place, with small, frequent pulse and cold extremities, and the child dies in from four to eight days from the onset of the attack. The temperature may remain normal, or rise several degrees, especially when there is peritonitis, till it falls at the period of collapse. The more complete the strangulation of the bowel the more rapid is the course of the case.

Now, abdominal pain, vomiting, constipation, and the passage of blood from the anus, coming on in a previously healthy child, are practically almost pathognomonic of intussusception, and the diagnosis is assured by the discovery of a tumour by palpation, especially in the right iliac region, or in the rectum. The following case from my note-books, which is typical of not a few, is a good example of what I have called the group of probable cases. A boy of 4 years old, previously quite well, had a fall on his head on the evening of July 16th, 1881, and seemed much collapsed soon afterwards. He was given some medicine, vomited, complained of much pain in abdomen, and passed a few loose stools. The vomiting continued, with great thirst, till admission into hospital the following midday. At 10 P.M. on the 17th vomiting was urgent, with signs of collapse; the abdomen was soft, but a small hardish lump, tender on pressure, was felt near the umbilicus. There had been no motion since admission. Nothing was felt in the rectum. A subcutaneous injection of $\frac{1}{24}$th grain of morphia was given, and iced milk in small quantities. After a sleep of several hours the vomiting returned. A simple enema (one pint) brought away a little greenish fluid and a few small fæcal lumps. In the afternoon of the 18th two pints of soap and water were slowly injected into the rectum, the boy being held with upraised buttocks, and the abdomen simultaneously manipulated by pressure directed from the groin towards the umbilicus. Much of the fluid was retained.

The child vomited but twice during the night and the next day, but the pulse remained very weak. On the evening of the 19th he had another morphia injection. After vomiting twice more on the 20th, and retaining some iced milk given by the mouth, he passed a solid but unformed stool in the evening; the lump was no longer felt, and he recovered quickly. This case may be put down by some to fæcal obstruction, but the *suddenness of its onset* and the unformed character of the first stool point strongly away from this explanation. The temperature was scarcely above normal throughout. In another instance, very similar to this, where recovery made the diagnosis doubtful, the return of the child to the hospital in a few days, and its death with all the classical symptoms, followed by a post-mortem, proved the existence of intussusception. I agree with Henoch that the absence of a palpable tumour is of but little diagnostic import, as the abdomen is frequently much distended when the case is first seen, and it is but seldom that anything can be early felt in the rectum. I have only been sure of this latter sign in two cases, both of which ended fatally. The tumour caused by accumulated fæces can usually be indented by pressure, and the case is characterised neither by sudden onset nor discharge of blood. Peritonitis very often causes symptoms of obstruction but not of intussusception; though the onset of vomiting may be sudden there is usually here early distension of the abdomen, with much tenderness and pyrexia.

Cases of sudden and repeated vomiting at the onset of acute febrile disease, with constipation, are occasionally diagnosed as obstruction. The two following instances which I have seen of pneumonia being taken, and in one case treated for obstruction, are of sufficient variety and importance to be quoted. The first was a boy of 6 years old, who was admitted to hospital with a history of repeated vomiting of yellow liquid for four days, beginning after a meal of boiled pork. The bowels had not acted for six days. Three days before admission he complained of pain with abdominal tenderness, but there was no distension; on the day before admission his vomit was said to "smell like motions." The next day the vomiting ceased. He had five enemata, by medical advice, with no result. On admission the temperature was over 102° F.; the abdomen was distended with some tenderness, but nothing was felt by palpation or rectal

exploration. The bowel was ineffectually inflated; there were signs of bronchitis but no pulmonary dulness. The next day the child vomited twice, but not fæcally; and the head was retracted. A gallon of tepid water was injected, retained for a while, and returned slightly discoloured. The child seemed to be dying. The day after admission abdominal section was performed; everything was found perfectly normal, and four ounces of liquid fæces were passed from the anus during the operation. Two days afterwards the child died with symptoms of peritonitis, and the post-mortem revealed recent peritonitis and enlarged mesenteric glands, but no stricture or intussusception. There was, however, double pneumonia, on one side in the stage of grey hepatisation.

The second case, a girl of 4½ years, was sent by medical advice to Westminster Hospital as intestinal obstruction for operation. She was said to have been well till two days before admission, when, in the afternoon, she looked pale and ill, went to sleep for an hour, and then woke up with abdominal pain and vomited. Very frequent vomiting, with pain in head and abdomen, continued till admission, vomiting following always on attempts to eat or drink. Between the attacks of vomiting the child was very drowsy. She had a slight cough which had lasted some months. Previous to this attack she had had some diarrhœa, but there had been no action of the bowels for two days before admission. When seen in hospital she was much exhausted; the temperature was 104·8°, pulse 138, respirations 40, and the face was flushed. Nothing was detected on examination of the chest, and no pain was complained of. Some acute febrile disease, probably pneumonia, was suspected from the temperature and general appearance, which was unlike that of abdominal mischief, and nothing was discovered by abdominal or rectal examination. A normal action was induced on the following day by an enema which was easily administered. The temperature and pulse remained high with no further action of the bowels; on the third day from admission signs of pneumonia at the left apex were first discovered, and at midnight on the next day there was a critical fall of temperature with sweating. The repeated vomiting in this case, which is exceptional in pneumonia, and the suddenness of its onset with abdominal pain, might fairly raise a suspicion at first of intestinal trouble,

but the condition of the child on admission rendered this diagnosis then extremely unlikely.

Prognosis and treatment.—A careful study of all the cases of intussusception which have come under my notice leads me to believe that the prognosis is generally grave when the diagnosis is certain, and especially when the invagination is felt in the rectum. Yet many cases recover with prompt treatment. My colleague, Mr. Parker, has made the valuable observation from his cases that absence, or slight amount, of blood-discharge from the anus, and, indeed, a short period of pain or vomiting at the outset, followed by a time of comparative comfort, are by no means necessarily of good import, as these conditions are often concurrent with rapid gangrene of the intestine leading to death in collapse; while a free discharge of blood signifies, by itself, incarceration rather than strangulation. On the other hand, I would say that such a condition as causes a free discharge of blood must, if persistent, soon become one of strangulation, and points to prompt remedial interference. It may, I think, be said that the cases which may recover from treatment are, first, those where with initial pain and vomiting and obstinate constipation with or without a tumour detectable through the walls, there is no discharge, or but slight, of blood from the anus, and no considerable abdominal distension; and next, those where there may be a notable amount of blood passed for a while, the abdomen being likewise undistended; while the gravest of all, and least likely to recover under any treatment, are those where, with obstinate constipation, the abdomen is distended, and the acute symptoms of pain, and perhaps vomiting as well, diminish early, or cease altogether. Constipation, therefore, of sudden onset, attended by pain or vomiting, in a previously healthy child, should always raise the suspicion of intussusception, and calls for early treatment, without waiting for that abdominal distension which even in the absence of any palpable tumour in many cases, may clinch the diagnosis. The discovery of a tumour or the discharge of blood would strengthen the diagnosis and emphasise the need of immediate relief. It is thus clear that treatment must often be begun before the diagnosis is settled; in waiting for certainty the chance of recovery may be lost. A definite case untreated of three days standing will generally die whatever is done, although in exceptional instances

there may be recovery. I leave out of consideration here the rare cases of chronic intussusception which generally end unfavourably, and the occasional instances, also, in later childhood, of recovery after sloughing away of the imprisoned gut.

When there is a reasonable suspicion of intussusception chloroform should be given, or opium or morphia cautiously administered till drowsiness be produced. The bowel should then be copiously and slowly filled with tepid water, the buttocks being well elevated by placing the child on an inclined plane, and the abdomen simultaneously manipulated by pressure directed towards the umbilicus. Several cases of undoubted nature including the presence of a palpable tumour have been in this way successfully reduced at the Children's Hospital. When the gut is gangrenous or very thin, the operation may cause perforation and rapidly ensuing death. This possible accident, however, in no way contraindicates the invariable necessity of having recourse with due care to this procedure. If this method fail, air may be very carefully injected; but after two unsuccessful trials at most with either liquid or gaseous injections abdominal section should at once be performed. At this stage, the treatment being surgical, I would but add that if reduction be impossible, or the gut in a very bad state, an artificial anus should be made, and gangrenous parts removed. In cases where a tumour can be felt in the rectum gentle attempts may at first be made at reduction by means of a sponge-tipped probang or bougie, but little is to be expected from this procedure. In older children, as pointed out by Dr. Eustace Smith, when there are very severe symptoms with much prostration, operation by section is generally contraindicated, as not only giving little hope of success, but also precluding what little chance there may be of cure by sloughing, which occasionally takes place even in cases apparently desperate. I have known, however, but of one such case, and of its after history I am ignorant.

FRACTURES OF THE LOWER JAW.

By MORTON SMALE.

A case that was sent to the Dental Department of the Hospital during the past year made it appear that a short paper on the treatment of fractures of the lower jaw might be of some interest.

It is impossible within the space at command to deal with all the varieties of fractures, simple, compound, and multiple, nor with the many seats of fracture in the course of the horizontal and ascending ramus of the lower jaw. It is intended in this paper to deal exclusively with the ordinary simple fracture such as a surgeon at any time may be called upon to treat, and as far as possible to suggest methods that will be within his reach. In another Report it may be possible to deal with some of the more complicated varieties; but in towns where there is to be found a really competent dentist it will be always advisable to seek his aid in such cases, his mechanical knowledge and skill being valuable adjuncts to that of the surgeon's.

The fractures with which it is intended to deal, then, are those that occur at any part of the horizontal ramus, usually compound as to the mouth, but simple, nevertheless, the usual seat being at or near the canine tooth; but the fracture may be in any part; and those cases where the fracture is situated behind the molar teeth in the neighbourhood of the angle, are best treated by a four-tailed bandage, the form and application of which are fully treated in all surgical manuals, the mouth being kept religiously closed during the whole process of healing, which should extend over a period of five to six weeks, the feeding being performed by fluids which can be readily passed into the mouth along the external side of the teeth, and allowed to get into the mouth round the back of all the teeth, or it can be injected with a curved nozzled enema or syringe.

The ordinary simple fracture should be treated if possible by

taking with a dentist's ordinary tray and wax a model of both the upper and lower jaw. Plaster of Paris mixed, after this mass has hardened, rather thin is then poured into these impressions and models made, the wax being removed from the plaster of Paris when dry by putting it in hot water; it will then be possible to divide the lower model with a saw at the seat of fracture, and by carefully articulating the masticating surfaces of the lower and upper teeth, thereby restore the models to their proper relative position (some paring of the two pieces of the lower model may be necessary). The two pieces of the lower model must then be united in a bed of plaister while they are in opposition to the upper jaw by means of the dental articulation.

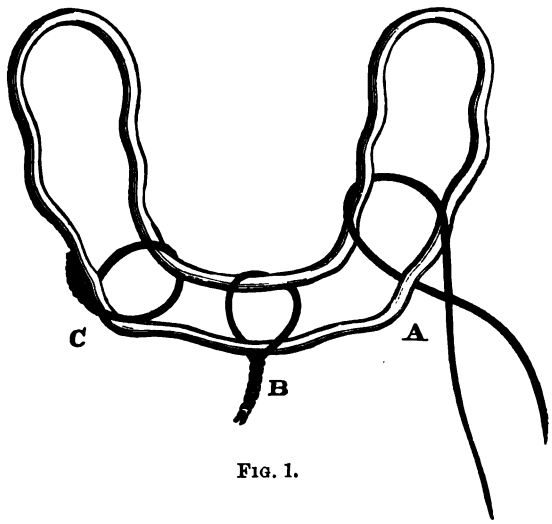

FIG. 1.

This will give a model of the mouth as it was before the fracture. It is then possible to make a wire splint to keep the fractured parts in position in the mouth as follows. (This splint was invented by Mr. Hammond during the Franco-German war, and has been named the Hammond splint.)

Take a long piece of thick iron wire or pianoforte wire and bend it to fit inside the teeth, then continue it round the back teeth and round the front of the teeth so that the teeth are all enclosed between the wires; the two ends of the wire must now be united, and this is the difficult part; if they be hooked together the ends would probably cut either the gum or cheek

unless the junction be made exactly behind the last molar. The best plan is to solder or braze the ends together; any tinker could do this if asked. The instrument makers might with advantage keep in stock a series of these splints already made, of various sizes, that could with very little bending be made to fit. Having made the splint, which when complete should be like fig. 1, it has to be fitted into the mouth, and for this purpose some pliers and forceps and some ordinary binding wire is necessary.

The splint being in place in the mouth each individual tooth

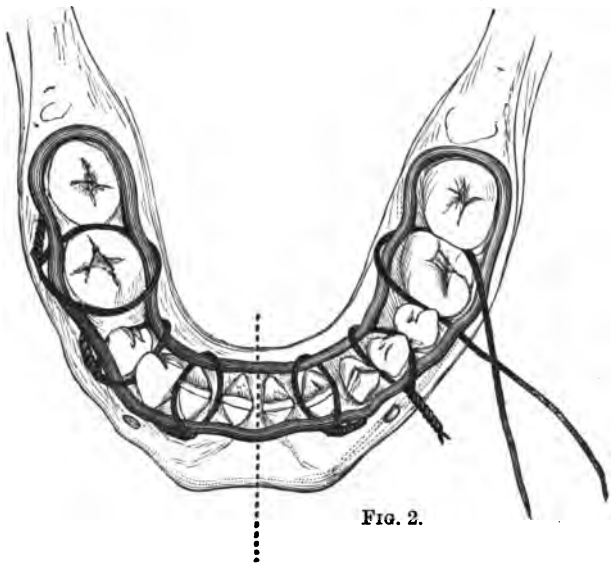

FIG. 2.

has to be attached to it with the binding wire. Between the necks of the teeth there is always a space; it is through this space that the binding wire is placed as follows. Start by passing the binding wire over the outside wire and under the inside one, and on returning it round the other side over the inside one and under the outside one; the two ends are then twisted (tightly enough to hold, but not tightly enough to break the wire) the ends cut off and bent out of the way between the teeth so that they do not cut the cheek. Each tooth having been treated in the same way, the jaw will be found to be quite firmly in position, so much so, indeed, that the patient is able to use it at once.

It is always advisable to look very carefully and see that a tooth has not slipped between the fragments.

There are cases in which this splint is not applicable, where, for instance, only a few teeth are standing; these are best treated by a cap which fits very roughly to the mouth; this lined with gutta-percha and fitted to the model, and then placed in the mouth. To keep this in place it is better either to tie it to the remaining teeth or to attach wings to it which come out of the angle of the mouth on either side, and to which a bandage can be attached to go under the chin, and keep the jaw into the splint (see cut). Several sizes in these splints might be kept in

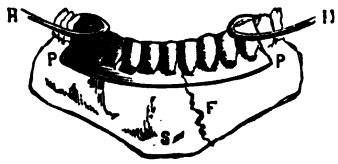

FIG. 3.

stock by the instrument makers for the use of surgeons. With these splints it is possible to do without a model when the fractured parts can be got into position. One of these caps can be filled with hot gutta-percha, the fracture being kept in position by an assistant. The splint can be put *in situ*, and both it and the jaw held until the gutta-percha is cold; this will be found a very simple method of treating a fracture for the surgeon, but not quite so pleasant for the patient as a wire splint.

I am indebted to Mr. Christopher Heath for the illustrations, he having kindly lent me the blocks.

NOTES ON THE GERMICIDAL ACTION OF THE BLOOD.

By Dr. HEBB.

It has frequently seemed to me to be convenient to regard the blood and other body juices in the light of cultivation media for the propagation of certain microbes which by their development give rise to certain phenomena, the sum total of such phenomena induced by any special micro-organism being a disease, say, enteric fever, scarlet fever, cholera, &c.

Now, on consideration, it appears to be very wonderful if a disease be the result of the action of microbes on the body why one patient dies, another recovers, and another, although exposed to the same poison, is not affected at all. Why, in fact, is one patient immune, why does another acquire immunity, and why does the third succumb?

For some time past attempts have been made to answer these questions (practically, however, they are one) by experimenting with certain microbes on the blood and other fluids of the body.

The experimentalists are divided into two schools, namely, those who feel assured that they have found the answer to the riddle in the corpuscular elements, and those who are only satisfied with replies from the plasma.

Thus a very curious position is arrived at: both sides are equally agreed about the blood, and each side is equally decided that it is only a part of the blood which is effective. The war-cry of the one side is phagocytosis, the other side emblazons its banner with the legend, germicidal action of blood serum.

There is no doubt that the doctrine of phagocytes is extremely taking. Phagocytosis, or the theory of phagocytes, as its author, M. E. Metschnikoff, calls it, was officially consecrated in 1887 (Annales de l'Institut Pasteur, vol. i., 1887), although it had been promulgated several years before (Virchow's Archiv,

1884, vol. xcvi. p. 177; vol. xcvii. p. 502). In the abstract it is simplicity itself. It merely consists in a cell catching a microbe, and then getting rid of it by a process of intracellular digestion. In itself, therefore, there is nothing wonderful about this, for the self-same struggle between low animal and vegetable existences is going on all over the universe. The small shapeless mass of animal protoplasm envelopes some small vegetable existence in its putty-like embrace, closes over it, then the outline of the vegetable organism grows dim and indistinct, and at last disappears altogether.

Under other conditions the same process goes on. Ranvier describes how the absorption of the myelin in damaged nerves is effected by amœboid cells in a manner analogous to the ingestion of fat or of particles of carmin by leucocytes after injection into the peritoneal sac, phenomena observed half a century ago by von Recklinghausen and Goodsir. So that apart from the special question at issue there is nothing at all strange or new in the fact that a single animal cell can incorporate and assimilate vegetable cells and also animal cells.

Now the cells called phagocytes act in exactly the same way; they can eat up both vegetable and animal microbes, and are not at all particular about the rights of personal liberty, for they are cytophagi as well as phagocytes, swallowing their smaller brethren when they come within reach. Their tastes, too, are either peculiar or catholic, for they absorb inorganic foreign bodies with the same freedom as they ingest a bacterium, and it is in this way that the particles of carbon are found studded throughout the pulmonary tissue and in the lymphatic glands. (Hence it suggests itself to me that the part these carbon-phagocytes play is not a selective one, in other words, they are merely irresponsible coal-heavers.)

The phagocyte is of two kinds, the small or microphage, and the large or macrophage. The microphage is a small, often polynucleated, cell, which has emigrated from the blood or lymphatic vessels, a leucocyte in fact. The macrophage is a large mononucleated cell almost always stationary. Between the two are various transition stages, from which it would seem that the macrophage is but a microphage in a high state of development. They are also stated to be derived from connective tissue elements, or from the epithelia of the pulmonary alveoli. It is most con-

venient, however, to regard the macrophage as a leucocyte, which having made its pile and become plethoric retires from active pursuits, although it still continues the habit of snapping up unconsidered trifles.

So the process is this: the leucocyte wanders out of a vessel, finds its way between the cells of the tissues, and perambulates, mayhap, even on to the free surface of the mucosa. Somewhere or other it comes across a foreign body, and having clasped it in its toils, tries to find its way back again, the process of digestion going on the while. Sometimes it keeps its prey all to itself, sometimes, however, it falls in the way of a macrophage, which makes a meal of the microphage and its partially digested dinner. This is the phagocyte's ordinary every-day life, a hum-drum sort of existence. When, however, the foreign bodies take the shape of pathogenic bacteria the state of affairs is greatly changed, although, of course, the method and principle remain the same.

In this case hunting is changed into fighting. Now, just imagine an attack in force on a given point by a bacterial army. Almost instantly the leucocytes hurry up, soon the tissue is overcrowded with reinforcements, and unless the enemy's battalions be too big, or the quality of the troops be too good, or they be too well armed, the march of the militant microbe is arrested. If, however, the battalions be too big (in other words, if the dose of the poison be too great), or if the enemy's troops be too well armed, or the phagocytes enervated by too long a peace (in other words, if the virus be too strong, or the organism weak or "predisposed"), then the microbe proceeds to settle down and colonise the country. But the phagocyte, though beaten at first, does not give in even then, far from it. He pulls himself together, and ejects the invader (period of recovery and convalescence). The victory is frequently marked by considerable changes, some for good, some for ill. The marks of the struggle may still be visible though the organism have recovered (variola, scarlet fever, &c.). It may be left in a condition which makes it an easier prey for subsequent attacks (*e.g.*, endocarditis). It may not only have destroyed certain parts, but colonies are still left which, though in a condition of repression, are ready to take up arms on the slightest provocation (*e.g.*, tuberculosis). But on the whole something has very frequently been gained, and that is immunity. The invaders may attack again, but this time the phagocytes are

on the alert; they have bought their experience and have learned how to tackle their old foe, and they make short work of him.

Such, in a few words, is the theory of phagocytes, a doctrine which has the double merit of being dramatic and Darwinesque.

Metschnikoff has been followed by a band of enthusiastic neopathologists, chiefly in France, who, while confirming their master's facts, have made no further discoveries of importance. Among these may be mentioned Hess (Virchow's Archiv, cix., 1887, p. 379); Tchistovitch (Annales de l'Institut Pasteur, iii., 1889, p. 337) on phagocytosis in the lungs; and Ruffer (Quarterly Journal of Microscopical Science, 1890, p. 481) on the phagocytes of the alimentary canal.

Yet despite its elegance, its appeal to the theatrical side of our nature, and its confirmation, it must not be supposed that the theory is accepted by everybody. Not only is the theory not accepted, but the facts on which it is founded are disputed. Thus some, while admitting the presence of bacteria within cells, claim that they are only devoured when dead, and hence incapable of offering any resistance. Others find that the bacteria disappear spontaneously about four hours after injection, and this without the intervention of phagocytes; while, according to another set of experimental critics, the disappearance of the bacteria is not due to their destruction by the blood, but to their habit of concealing themselves in the capillaries of various organs (Flügge, Grundriss der Hygiene, 1889, p. 487; Czaplewski, Centralbl. f. Klin. Medicin, 1888, p. 516; Lubarsch, Centralbl. f. Bakteriol. u. Parasitenk, vi. p. 486; Wyssokowitsch, Zeitschrift f. Hygiene, 1886, p. 1; Flügge, Mikro-organismen, 1886, p. 606).

As these objections strike at the roots of the phagocytic theory, Metschnikoff replies by giving details of further experiments, more confirmatory than ever of his doctrine (Etudes sur l'Immunité, Annales de l'Institut Pasteur, 1890, p. 65). Herein he shows that phagocytes, macrophages as well as microphages, not only take up bacteria while still alive, but that these have retained their virulence.

But the most plausible objection to phagocytosis is drawn from the germicidal action of the blood plasma. The experimental pathologists who hold this view proceed by freeing fresh blood from its corpuscles and then noting the diminution in the

number of bacteria found in the cultivation medium at various intervals of time after inoculation. At first there is a diminution in the number of bacteria. On this fact the experimental pathologist builds the superstructure, the germicidal action of blood plasma. One of the first of these was Nuttall (Zeitschrift f. Hygiene, iv. 353). The experiments on the "germicidal influence of the animal body" showed that the efficient agent was the blood plasma, and also that the bacteria were dead before incorporation. In the course of this investigation it was found that the germicidal power became used up in the process of destruction, that it diminished with lapse of time, and at temperatures above 50°.

Another was Nissen (Zeitschr. f. Hygiene, vi. p. 487), who, experimenting with a large number of micro-organisms, found that while some would not live in blood, others throve well, the pathogenic and saprophytic microbes being affected in an analogous manner, some kinds of each were destroyed, and some were not. Among the pathogenic which were easily vanquished were cholera, anthrax and typhoid, while among those which throve well were certain exciters of suppuration, such as Staphylococcus pyogenes aureus, St. pyogenes albus, and Streptococcus erysipelatis. This author further found that when a large number of micro-organisms was injected (overdose of poison) the germicidal action was lost. In order to determine the principal agent in the germicidal action, blood was rendered incoagulable by adding peptone or sulphate of magnesia, and blood serum carefully freed from corpuscles also was used. Both the serum and the peptonised blood showed germicidal properties, but the salinated medium did not. From the results of these experiments it was concluded that the serum possesses the chief germicidal action.

But the most prominent opponent of Metschnikoff and the most ardent supporter of the germicidal virtues of the plasma is Buchner, who (Centralbl. f. Bakteriol. u. Parasitenk, vols. v. p. 817, and vi. p. 1) found that fresh blood which had been deprived of its corpuscles rapidly killed bacteria. The results, however, were different for different kinds of serum, and he also noted that the germicidal capacity was limited. In conjunction with Voit, Sittmann, and Orthenberger (Archiv f. Hygiene, x. 1890, pp. 84, 173), Buchner has made further progress in

researching the "germicidal action of the blood and blood serum." By these experiments the germicidal action of the plasma is not only demonstrated, but it is further shown that it is the serum which is possessed of this property, and still further that this function is clearly associated with albumen.

Other observers who have followed in the same line are Lubarsch, Charrin, and Roger, Petruschky and Fodor.

This last observer pursues the subject still further (Centralbl. f. Bakteriol. u. Parasitenk, vii., 1890, p. 754), and concludes from his experiments that there is a direct connection between the alkalinity of the blood and its germicidal action. And as this latter was found to be augmented by the addition of alkalies, hopes are thrown out that eventually immunity may be acquired by the discovery of some procedure for saturating the blood with alkalies.

Prudden, in pursuing the same object (Medical Record, January 25th, 1890), experimented with the bacillus of typhoid in ascitic and hydrocele fluids, and concludes from the results he obtained that, like blood, fresh non-inflammatory transudates possess a marked germicidal power which is in some way dependent on their albuminoid constituents.

But besides the corpuscles, the plasma, and fluids derived directly from the blood, fluids indirectly derived, for example milk, also possess a germicidal influence. Milk which has been examined by Fokker (Fortschritt d. Medicin, viii. 7), if boiled and inoculated with the lactic acid bacillus is found to coagulate within 24 hours, if unboiled in two to four days. Just as in the case of the blood, there was at first a decrease in the number of bacteria, afterwards followed by the usual increase.

Hence it may be seen that the antiphagocytists, whether they start on the blood plasma or on its direct or indirect derivatives, are quite agreed that all these fluids possess a germicidal action, and that this power is the property of the plasma alone, and that the corpuscles are not endowed therewith. And that is how the matter stands at present.

To the outside observer it would seem that both parties had proved, experimentally of course, their point, and that it might be advisable to amalgamate the results, or to treat the subject from a slightly different standpoint. In other words, why not assume the unity of the blood? Why dissociate the corpuscles

from the plasma? Why not consider the blood as one and indivisible? If any useful view of the germicidal action of the blood is ever obtained, this, I take it, will in all probability be from the blood as a whole, and not from any single constituent.

However, it is interesting and comforting to learn that it is experimentally certain we carry about with us either a large number of active disinfecters, or a good quantity of passive disinfecting principle by which a pathogenic virus, unless too much in quantity or too strong in quality, is sooner or later abolished.

Notwithstanding all this, the great question, Why does one attack usually confer immunity? is still unanswered, and until it is, it will be more convenient, and certainly much safer, to regard the blood as a cultivation medium than to endow it with germicidal virtues, lest overweening confidence in this function betray us into the power of the microbe.

Though the foregoing may be regarded as a fair summing up of the case of corpuscles *versus* plasma, there are other aspects of the blood or immunity problem. These are either details, though important ones, or views of the question regarded from a different standpoint. As an example of the latter may be mentioned the researches of Hankin, who, arguing up from intracellular digestion, arrives at the conclusion that the germicidal, or, as he prefers to term it, bactericidal, influence may be due to the action of a ferment circulating in the blood, say pepsin or trypsin. Although his hypothesis is plausible and elegant, the results of his experiments for conferring immunity by fermenting the blood with pepsin or trypsin are so far not very encouraging (British Association, *Times*, September 6th, 1890). As showing to what extent phagocytosis is now adopted, it should be mentioned that Hankin alludes to phagocytes in a way that proves their action is taken for granted, though the prominent position is, of course, assigned to the ferment.

This author had previously contributed to the blood question (Proc. Roy. Soc., May 22nd, 1890) by describing a bacteria-killing globulin. This globulin was discovered by Halliburton (Journal Physiol., ix., 1888, 229), who called it cell globulin β, and identified it with fibrin ferment. Hankin's experiments,

however, went to show that this globulin possessed germicidal properties, and these served to distinguish it from fibrin ferment.

As illustrating how a detail may become a prominent factor, I will here allude to the concentration of the medium. The small controversy on this subject is interesting, not only in itself, but also as an example of the liability of the scientific experimental mind to quite an ordinary human frailty. The one side, finding that the other really had some facts in its favour, proceeded to explain these away by hinting that the germicidal action was due to the concentration of the medium. By concentration of the medium an inspissation or increased density thereof is meant. Now in the case under consideration the concentration of the juices of the body is assumed to be greater than that of the artificial experimental media. Thus taunted, the defender of the germicidal action of the plasma promptly accepts the challenge, and retorts that he has previously shown that serum when heated to 55° loses its germicidal property while maintaining its concentration, and further shows that on increasing the concentration by addition of sugar or pepton no practical difference is observable in the result (Buchner, Centralbl. f. Bakteriol. u. Parasitenk, viii., 1890, 65; Metschnikoff, Annales de l'Institut Pasteur, 1889, p. 664).

Yet notwithstanding these experimental facts the opposite conclusion has been arrived at, also experimentally, by Eschenhagen (Berichte Verhandl. Sächs. Gesell. Wiss., Leipzig, 1890, 343), who found that concentration of the nutritive medium did exert an inhibitive influence on the growth of fungi. The latter conclusion is probably the correct one, inasmuch as it agrees with the jam-making experiments of Messrs. Crosse & Blackwell, and others. This remark is, however, open to the criticism that jam is boiled, but then so are most things in a scientific experiment.

Whatever any one may think of the value of the concentration of the cultivation medium, natural or artificial, the objection illustrates a weakness of human nature. This weakness, as I hinted before, though shared in by the experimental mind, is more commonly found in the sex usually placed above senior wranglers. The frailty consists in the constant habit of giving an uncalled for explanation to an otherwise unanswerable criticism. Like the ladies, the phagocytist, brought to task by the plasmatist, proceeds to offer an explanation of how it all

happened. But it may be asked, Why trouble about the doings of other people if satisfied with our own position?

Quite so, and considering the whole state of the question it seems to me weak on the part of the phagocytist, for he thus practically confesses that his doctrine is the offspring of an agnostic mind, which, clinging to the pendulum of suspense, sways to and fro over the sea of doubt between the shelving shore of orthodoxy and the rugged-looking banks of heterodoxy.

It would be impossible to pass away from the subject without referring to the hæmatozoa of malaria. To a Frenchman (Laveran, Traité des Fièvres palustres, Paris, 1883), are due the honour and glory of first discovering and describing these parasites the existence of which has since been abundantly verified by Richard, Marchiafava, Celli, Osler, Canalis, Golgi, and others (Archiv Italien Biol., viii. 1887, p. 131, also p. 154; also vol. xiii., 1890, p. 262; British Medical Journal, March 12th, 1887).

Though numerous descriptions of these small animals have been given, it will be sufficient here to say that they are amœbiform in character, and are roughly divisible into two kinds, the amœboid and the crescentiform. They almost always contain pigment granules, and usually inhabit the red disks or the plasma, being only occasionally observed in the white corpuscles. One statement recently made about them will, if it be confirmed, be as important as it is interesting. It is said that the amœboid variety is associated with the regular type of malaria, while the crescent shape is found in the irregular types. In their juvenile condition they are indistinguishable (Grassi and Feletti, Centralbl. f. Bakteriol. u. Parasitenk, viii., 1890, 396).

Now, much in favour of the actual existence of these parasitic protozoa is that there is no Egyptian or Etruscan mystery about the business. No special apparatus or procedure is required for demonstrating them. The only necessary articles are a microscope, a slide, a cover-glass, and a needle, though, of course, the blood may be fixed and stained in the usual manner. Moreover, the upholders are constantly receiving the adhesion of some of their former opponents, and one of those who has

gone over to the true faith, after acknowledging his errors, exclaims against "the folly of a sceptism based on theoretical considerations and of preconceived notions drawn from a limited experience." Such confidence would seem capable of removing Himalayas of doubt.

Yet, notwithstanding these statements, though accompanied and supported by numerous diagrammatic illustrations, equally distinguished investigators, physiological as well as pathological, while admitting the appearances observed in malarious blood, are of opinion that these appearances are merely the result of alterations and changes in the red corpuscles, degenerations, in fact, due of course to the malarial virus. One of these, who has written a large work on the blood, states that the appearances described may be found any day in ordinary blood; although he frankly admits that he is not personally conversant with the appearances of malaria blood. Mosso makes a similar statement; but another distinguished observer, Tommasi-Crudeli, does not mince the matter. To him the plasmodium is a fraud. "*Questo preteso parasita, chiamato da alcuni, plasmodium malariæ, e poi da Metchnikoff coccidium malariæ, non esiste.*" (Hayem Du Sang, Paris, 2nd edition, pp. 347—9; Archiv Ital. Biol., viii., 1887, p. 252; Atti reale Accad. dei Lincei, 1888, pp. 305—8).

Such a statement verges on scientific Billingsgate, and should be received with a considerable amount of salt, for the writer, in conjunction with Klebs, palmed off on the unwary bacteriological world, at a time when it was somewhat excitable about microbes, a bacillus malariæ. Owing to the appearance of the more juvenile candidate, and its warm reception, the poor bacillus malariæ has had its nose put out of joint, and its sponsor, who still supports it, seems correspondingly exasperated.

To keep the points of such a scientific squabble as this well before the view is extremely serviceable; but the plasmodium becomes still more interesting when we find that phagocytosis again crops up, for the aid of the phagocyte is required, or at least not objected to. It is convenient to have its support to fall back upon; the presence of the pigment granules in the spleen, liver, and bone marrow is then easily explicable. The friendly phagocytes have been at work again. They have returned from the war-path and have deposited their trophies in

their favourite haunts. The dried scalps of their foes are hung up in the wigwams, and then all is peace for a time.

In making these short notes my intention was to show that there were other views anent the germicidal action of the blood besides that of phagocytosis. Everybody is now raving of phagocytes, they have even got into the lay papers, and it was to caution those who might happen to read these lines against being drawn into the supposition that cultivated and well-educated leucocytes were solely responsible for inherited or acquired immunity from infectious and contagious disease. The examination of the blood is as yet in its earliest infancy; what little is known about our vital fluid is somewhat meagre after all. If we confine ourselves to the mere examination of the corpuscles, a very short experience is sufficient to show us that this examination is beset with more difficulties than appear at first sight; while if we essay the plasma, the methods of examination at present used only serve to render evident certain physico-chemical changes, the result of altered conditions and environment. The blood, as it is examined, is no longer blood; it is something else. If we do not care for the trouble of examining, this same conclusion can easily be arrived at by consulting any work specially devoted to the blood. Though these difficulties may not appear very satisfactory at first sight, a little reflection will reconcile us to the fact, for we shall be led to the conclusion that there is more in the blood than the text-books of physiology yet contain about it. Nay, it almost raises a pleasurable excitement to find that there is still a large playing-field wherein physiology and pathology may disport themselves. Yes, I quite agree with the opening words of the preface to Hayem's monograph on the blood, "*L'avenir appartient à l'hématologie.*" There is a big future before it.

While enumerating the foregoing speculations on the germicidal action of the blood and their bearings on immunity, I am not professing to make the list complete; but as far as the general aspect of the question is concerned they will be sufficient not only to show the different drifts of men's minds, when they

come to deal with the question, but also the difficulties and the vastness of the subject. On no single theory can perfect reliance be placed for explaining the facts made out by clinical observation or experimental research; but if I were called on to express an opinion, I would say that, taking our present knowledge into consideration, my predilections would lead me to adopt a view that acquired immunity from infectious disease was chiefly the result of a fermentation, which by altering the constitution of the blood and tissues, unfitted them, at any rate for a certain period, to be a cultivation or fermentation medium. Such a hypothesis, and indirectly it includes inherited immunity, practically means that microbes cannot develope in an unsuitable medium, cannot exert their pathogenic function, for they have nothing to live on, makes in fact any distressingly active interference on the part of the blood unnecessary, and if it be neither elegant nor dramatic, it has the advantage of being convenient.

REPORTS OF SPECIAL CASES.

FOUR CASES OF INTEREST.

REPORTED BY W. W. POWELL.

A CASE OF SEVERE HÆMOPTYSIS IN ENTERIC FEVER.

FRANK S—, æt. 21, bricklayer, was admitted into Hallett Ward under Dr. Sturges, on May 8th, suffering from enteric fever. There were indications that the attack would be a severe one, and the patient was already (9th day) apathetic and suffering from much diarrhœa. The abdomen was slightly distended, and together with the back was nearly covered with typical spots. The usual tenderness on pressure over the right iliac fossa was complained of. The tongue brown at the tip, and the pulse soft and dichrotic. There was slight cough, but the chest was perfectly clear on auscultation, and the other physical signs were those of a healthy chest. The temperature ranged between 102° and 104° F. Two days after admission about an ounce and a half of bright frothy blood was expelled from the lung, and the following afternoon the hæmoptysis was most profuse, a pint of bright blood being coughed up in about an hour. Hypodermic injections of ergotine had the desired effect in arresting the hæmorrhage. Slightly impaired resonance with obscure crepitations were now detected over the apex of the right lung. These signs, however, soon disappeared. Laryngoscopic examination of the larynx revealed nothing abnormal. There was no phthisical taint according to the patient's personal and family histories, and he had never previously suffered from hæmoptysis. The recovery, after an attack of unusual severity complicated by many subcutaneous abscesses, was rapid and complete, the patient,

after a couple of months' holiday, presenting the picture of health and weighing fourteen stone, as against nine stone at the beginning of convalescence. The chest physical signs were perfectly healthy and there was no cough.

CARCINOMA OF THE SIGMOID FLEXURE IN A YOUNG WOMAN.

Emma S—, æt. 23, housemaid, was admitted under Mr. Davy into Percy Ward on September 22nd, suffering from constipation, abdominal pain, and vomiting, and having a tumour in the left ilio-lumbar region. The patient, who looked very pale and ill, had remained at her work until four days before admission, when she was compelled, through pain and weakness, to take to her bed. About four months ago, having been previously in her usual good health, a pain was first noticed in the left side of the abdomen. It was of a dull, aching character, with occasionally colic superadded. These symptoms persisted, and gradually grew worse. Constipation was not complained of, and indeed was not a sufficiently prominent symptom to attract notice until near the end. Attacks of vomiting commenced about two months ago, when she was sent into the country for a change. After a month's holiday, during which time there was occasionally severe vomiting, she returned to her work feeling much better in health and free from sickness. This period of comparative immunity from distressing symptoms did not, however, last long, for when she came under observation a week or two ago vomiting and pain had returned with increased severity. An abdominal tumour was now discovered, and constipation, alternating with diarrhœa, was complained of, and the patient was recommended to come into hospital. When admitted, the patient, who was very pale and looked extremely ill, frequently vomited the brownish green contents of the upper part of the intestinal tract. On palpating the abdomen, which was slightly distended, a resistent tumour was easily felt occupying the left ilio-lumbar region, and extending round to the spinal muscles behind. Whatever its nature, its presence was evidently the cause of the severe gastro-intestinal symptoms, and treatment

was naturally directed for the relief of the almost complete obstruction that existed. Large enemata of soap and warm water were administered by means of a long rectal tube, and although these were frequently repeated, nothing came away, and no good resulted. Belladonna fomentations were applied to the abdomen, and opium and belladonna pills were given at short intervals. In two or three days stercoraceous vomiting supervened, and the advisability of an operation was discussed, but the patient was so feeble that any such interference was considered unjustifiable under the circumstances. The patient rapidly grew worse, and sank at the end of the sixth day after admission.

Post-mortem a large neoplasmic mass was found involving the commencement of the sigmoid flexure of the colon, and having its starting point in the gut, which was strictured by the growth. The adjacent organs were healthy. The glands were not apparently enlarged. The colon was greatly distended above the growth, and contained a large quantity of semi-fluid fæcal matter.

Microscopical examination by Dr. Hebb. The tumour is a columnar epithelioma, forming follicles, which contain colloid matter. The colloid matter is evidently a secretion from the epithelia, and is not due to a degeneration of the cells themselves.

CARCINOMA OF THE MALE BREAST.

William B—, æt. 62, an old soldier, was admitted on August 7th into St. Matthew Ward under Mr. Cowell, suffering from a tumour of the right breast. There was no family history of malignant disease or tumours of any kind. Patient's left mammary gland was scarcely palpable, and, according to his account, the right was in a similar undeveloped condition before the commencement of the present growth. There was no history of a blow. About two years ago the patient, whilst washing himself, noticed a small lump, about the size of a marble, just below and external to the right nipple. It was hard, smooth, and freely movable. Six months after this a medical man was

consulted, who advised leaving it alone, its presence occasioning no pain or inconvenience. It was now about the size of a walnut, and still smooth, hard, and movable. In another six months, however, it had increased considerably in bulk, and during the last three months its growth has been very rapid, the skin becoming blue in parts, and the tumour softening. There was now, at times, much pain of a gnawing character.

On admission, an oblong tumour, adherent to the skin, but movable over the pectoral muscle, about 4 inches long, 3 inches broad, and very prominent (2¼ inches), with steep, almost perpendicular, sides, extended downwards and outwards across the right mammary region, and presenting, over the lower two-thirds, where the skin was of a purplish colour, a bulging and bossy appearance. Over this area fluctuation could be obtained on palpation. The upper or more solid portion was semi-elastic to the feel, the skin here being of the normal colour, and showing enlarged superficial veins. The nipple was normal in appearance, and there were no enlarged axillary glands to be made out. Patient, who is a spare man, says that he has not been losing flesh.

On the 12th the tumour was excised with antiseptic precautions by Mr. Cowell, the patient being anæsthetised with ether. An incision, tapering to a point at each end, was made along the base of the tumour, the skin covering the latter being removed with the growth. A few touches of the scalpel, together with slight traction, sufficed to raise the base from the pectoral fascia, which was not implicated. Four or five arteries having been ligatured, and the wound cleansed with carbolic lotion, the edges of the skin were brought into apposition, and without tension, were secured by the interrupted silver suture, no drainage tube being used. The wound was dressed with cyanide gauze and iodoform wool, and the arm fixed to the side by a few turns of bandage in the usual way. The dressings were changed on the third day, and again on the sixth, when the stitches were removed, the wound having healed by first intention. On the tenth day the man was discharged, feeling quite well, and presenting only an even linear scar where previously a large tumour had disfigured the pectoral outline. Six weeks afterwards the patient came to show himself. He looked remarkably well and had gained in weight.

The tumour, on section, was found to be firmly adherent to the skin, and presenting a smaller solid portion (the superior *in situ*) and a larger cystic portion. The cut surface of the solid part was of a dirty white colour, firm in consistence, of fibrous appearance, covered with minute spots of a lighter shade, and yielded a "juice" on scraping. The cysts, of which there were four or five, were imperfectly separated from each other, and were filled with a discoloured grumous-looking material consisting of broken down tissue and altered blood.

The microscopical examination showed the usual fibrous stroma, forming alveoli, which were filled with nucleated epithelial cells without intercellular substance.

SECONDARY CARCINOMA OF THE SCAPULA.

Charles L—, æt. 60, was admitted into St. Luke Ward under Mr. Macnamara on September 24th, suffering from a tumour of the left scapula, supposed to be an osteo-sarcoma. He also complained of inability to take solid food. The history was this: about five months ago the patient consulted a medical man for persistent slight difficulty in swallowing. For some time previously there had been occasional dysphagia, but very little notice was taken of it. He was told that he had a stricture of the œsophagus, and was recommended to attend at a throat hospital, which he did for some time, without, however, obtaining relief. There was now loss of appetite and vomiting on attempting to take solids, the food giving the sensation of stopping at the upper part of the gullet. About this time, and three months ago, a small lump was first noticed attached to the lower angle of the left scapula. It caused no pain or inconvenience, and, growing slowly at first, has been rapidly increasing in size lately, becoming also a little painful. The throat symptoms have persisted, and only liquids can now be taken. Blood has never been noticed in the vomit. There is no syphilitic history, and no family history of cancer. Patient states that he is rapidly getting thinner, due, he thinks, to his inability to take solid food. There are no physical signs of

aneurysm. On examination, nothing abnormal could be detected in the epigastrium, and there were no enlarged cervical glands. Laryngoscopic examination revealed nothing except a little redness and swelling about the left arytenoid cartilage. The œsophageal bougie has been purposely avoided and the diagnosis of œsophageal stricture rests on symptomatic evidence only. It has been suggested that the scapular growth may be a secondary carcinoma, the primary presumably being in the œsophagus, and causing the dysphagia. The treatment, however, in any case being the same—that of removal—no attempt was made at confirming the latter view by unnecessarily probing the gullet. The tumour, which was hemispherical in shape, about the size of half a cricket ball, and with a smooth, even surface, was incorporated with the inferior half or two-thirds of the infraspinous portion of the scapula. It was firm to the feel, having slight elasticity, and the skin and subcutaneous tissues were freely movable over it. There were no enlarged lymphatic glands. The patient having been anæsthetised with ether, and all antiseptic precautions observed, a T-shaped incision was made through the skin and subcutaneous tissues exposing the tumour partially covered by the muscles attached to the back of that part of the scapula. One or two vessels having been secured, the latissimus dorsi pulled downwards, and the other muscles divided or retracted, the scapula with the growth was easily lifted from the thoracic wall by placing the fingers under the inferior angle. The bone was now cleared of the soft structures, and sawn across a little below the spine, when, after the division of the muscles connected with this portion of the shoulder blade, the mass was removed. The bleeding vessels having been ligatured and the wound cleansed, the flaps of skin were brought into apposition and secured with the interrupted silver suture, a small drainage tube being placed in the inferior angle of the wound. Cyanide gauze and iodoform wool were used for dressing, and after the arm had been fixed to the side by a few turns of the roller, the patient was replaced in bed. The dressings were changed the same evening and again the following morning, there being rather free oozing of blood and serum. The patient made an uninterrupted recovery from the operation, the wound healing by the first intention, and he was discharged on the fourteenth day, feeling better than when

admitted, and with voluntary movements of the arm almost painless.

The tumour, which on section, was found to be more or less perfectly encapsuled, was greyish-white in colour, fairly firm to the feel, uniform in appearance, without cupping and yielding the so-called "cancer juice" on scraping. The remains of the scapula, reduced to a number of small spicules, occupied a plane of the growth about half an inch in thickness near its deep surface. At first, judging from this portion, the tumour was suspected to be an ossifying sarcoma, but closer observation revealed its true nature.

Microscopical examination by Dr. Hebb.—The tumour is evidently malignant. It has a fibrous stroma, forming alveoli, which are packed with indifferent cells, without intercellular substance, and each cell having a large oval nucleus and small amount of extranuclear protoplasm. In the centre of many of the alveoli are circular areas of degeneration, and there are besides large numbers of fibres, apparently fibrous tissue, which have undergone the so-called hyaline degeneration. It is probably, therefore, secondary carcinoma.

ELEVEN MEDICAL CASES OF INTEREST:

THEIR CLINICAL AND PATHOLOGICAL HISTORY.

By Dr. HEBB and Dr. WILLS.

Case 1. *Leucocythæmia* (*Leukhæmia*).*—Samuel R—, æt. 63, a gardener, was admitted under Dr. Allchin, on May 1st, 1889.

Twenty years ago, when in the army, he had had "inflammation" in the left hypochondrium, for which he was treated in Devonport Hospital for four months. Shortly after this he caught cold, and was seized with shivering fits, alternating with heat and faintness, the doctor telling him he had "ague." The attack lasted one night, and never recurred. Scarlet fever and smallpox when a boy.

In August, 1888, he noticed a swelling in the left hypochondrium, and he suffered from giddiness and headache. At Christmas time he had a severe attack of epistaxis, lasting off and on for two days. He had noticed himself getting paler and thinner for about three years.

On admission he was noted as being emaciated and anæmic. Pulse 80, somewhat irregular in rhythm. Apex beat feeble, scarcely perceptible; a loud, rough apex systolic murmur conducted all over the præcordia and towards the base. Lungs showed some emphysema. The spleen was considerably enlarged, extending to the level of the anterior iliac spine, and slightly to the right of the middle line at the umbilicus, surface smooth and hard. No tenderness. No increase in the area of hepatic dulness. There is some fulness of the superficial abdominal veins, especially on the right side. No ascites. No enlargement of lymphatic glands anywhere.

The blood was appreciably pale, and examined by the hæmocytometer, gave red corpuscles 2,000,000 per c.m., or ·40 of

* Post-mortem Book, vol. v., p. 264.

normal; leucocytes 1—12 red, or 50 times the normal number. The red corpuscles were smaller and paler than normal.

During the month of June he improved 4¼ lbs. in weight, notwithstanding an attack of diarrhœa, and also some dry pleurisy on the right side.

On June 22nd the blood gave with the hæmocytometer, red corpuscles, 2,470,000 per c.m., or ·49 of normal; leucocytes, 1—7 red, or 86 times the normal number, taking in each case 1—600 as the normal ratio between red corpuscles and leucocytes.

On July 10th he had some swelling and redness of the knuckles of the right hand, which soon passed off, and on the 19th slight hæmoptysis. He went out from the hospital on August 10th, feeling and appearing much better. When discharged his urine contained ⅕ albumen, from which it had been free on admission.

He was readmitted on October 9th, 1889. He stated that a fortnight after leaving the hospital he began to get much worse. He had had three attacks of epistaxis, which required plugging to arrest them. He had lost flesh, and his dyspnœa and palpitation had got worse. He had also suffered a good deal from cough and expectoration.

On October 11th he was noted as being paler and more emaciated than before. Spleen much enlarged since previous report, extending well below iliac spine, and displacing the umbilicus considerably to the right. No enlargement of lymphatic glands. Other physical signs much the same. No albumen in the urine. He rapidly got worse and died on October 16th.

Post-mortem examination was made by Dr. Allchin seventeen hours after death.

Body very anæmic, very slight œdema of feet and ankles. Moderate rigor mortis. No enlargement of superficial lymphatic glands. Some prominence of superficial abdominal veins. Rib muscles pale. Rib cartilages uncalcified. Several ounces of clear fluid and serum in each pleural cavity. A few old adhesions over right lung posteriorly. About 4 ozs. of similar fluid in pericardial cavity; about ½ pint in peritoneal.

The blood is of a somewhat grumous, chocolate appearance. The clots are long, and escape from the cut vessels in an ex-

tremely branched form. They are soft, pale, and chocolate in appearance. All the cavities of the heart are much distended with clots of a similar description. Heart 1 lb. 1¼ oz. Circumference of tricuspid 5 inches, of mitral 4¼ inches. The outer cusp of latter is much thickened, puckered, and also perforated. Its chordæ. tend. are enormously thickened, short, and very hard. And attached to the edge of this cusp are a few fibrinous flakes. Aortic cusps are much thickened, with extensive calcareous deposits in the sinuses. The orifices of the coronary vessels are patent, and there is little or no atheroma of aorta beyond sinuses. Aorta very elastic and healthy-looking. Heart substance pale, muscular substance visible to extreme apex of right ventricle. Average thickness of left ventricle = ½ inch.

Left lung crepitant throughout. A quantity of frothy serum escaped on section. Right lung crepitant to extreme base. Pleura over lower part is much thickened and scar-like, and the immediately adjacent portion of lung tissue is more fibrous than normal. Mediastinal glands somewhat enlarged, hard, and in part deeply pigmented.

Spleen 6 lbs. 3 ozs.; 12 inches long, 8 inches wide. Very slightly adherent to adjacent parts. Capsule moderately thickened. Surface slightly rough, and mottled with lighter patches beneath the capsule. On section uniformly pale, somewhat grumous-looking, and to some extent homogeneous and glistening, suggestive of lardaceous degeneration. No infarcts.

Liver 8 lbs. 4¼ ozs., very large; somewhat hard on section, with pale mottling. Gall bladder full.

Mesenteric glands somewhat enlarged, and present on section a typical pallid, soft appearance.

Suprarenals somewhat enlarged, otherwise normal.

Left kidney very pale, studded with cysts in size from pin's head to large pea. Capsule considerably adherent. Cortex considerably diminished, pyramids in places reaching almost to surface, smooth and glistening, again suggestive of lardaceous disease. Weight 4¾ ozs. Right kidney, 6 ozs.

Cancellous portion of ribs and sternum remarkably dry and dead looking. A small quantity of grey marrow only exuding.

Microscopical examination.—Sections of the spleen do not show any marked excess of fibrous tissue, but are characterised

by the exceeding number of large lymphoid cells, by the absence of the usual indications of the whereabouts of the malpighian bodies, and by the very small number of red corpuscles. Indeed this spleen bears a very strong general resemblance to a lymphatic gland in a condition of hyperplasia, and is not at all unlike the lymphatic glands in the lymphadenoma case. (See case 2.)

The liver sections show dilated vessels filled with lymphoid cells, and which in places form large aggregations. When examined under a high power ($\frac{1}{6}$ apochromatic) these lymphoid cells are found to be elements with large, usually single, spheroidal nuclei, and a small but quite distinct amount of perinuclear protoplasm. The nucleus stains deeply blue with logwood, while the plasma is best seen after colouring it with a red aniline dye. From their shape, size, and general appearance we are inclined to think that they bear little resemblance to a white blood corpuscle.

CASE 2. *Lymphadenoma (Hodgkin's Disease, Pseudo-leukhæmia).**—John L—, æt. 10, a schoolboy, was admitted into Burdett Ward, under Dr. Donkin, on October 8th, 1889.

He had measles æt. 2, and whooping cough æt. 3. No other illness. No history of ague or any exposure to its influence. He had lived up to nine months before admission in a very damp house, and he was often wet through and insufficiently fed.

He was in good health until the beginning of September, 1889, with the exception of a slight cough for about nine months, when he began to suffer from pains in the chest and shortness of breath, and his cough got worse. He had one attack of shivering at the commencement. He lost flesh and got much paler.

On admission he was pale and somewhat emaciated, perspiring freely about the head. Respirations 32. The veins were marked over the front of the chest, especially under the left clavicle. Resonance was good all over the front and back of the chest. Numerous moist râles all over both chests. Heart sounds normal. Apex in normal position. Abdomen considerably distended, no evidence of fluid, but the spleen could be felt to be much enlarged, reaching down to the crest of the ilium and to the right to within 1 inch of the umbilicus; upwards the dulness extended

* Post-mortem Book, vol. v., p. 390.

to the seventh rib. The blood examined with the hæmocytometer showed a deficiency of red corpuscles equal to half the normal, but no increase of leucocytes. Many of the red corpuscles were smaller than normal. During the next few weeks he gradually got worse. His breathing became more laboured, and he could not lie down. His pallor, if anything, became greater. About the middle of November there was found to be an area of dulness behind the upper part of the sternum, extending about 2 inches to the left of it at the level of second costal cartilage. Over this area bronchial breathing could be heard. In December the glands in the triangles of the neck became enlarged, and a few weeks later those in the axillæ and groins were felt to be shotty, but they never became much enlarged.

The evidence of enlargement of the mediastinal glands became greater, the bronchial breathing was heard at both apices and at the left base, accompanied by numerous moist sounds. His respirations became more frequent, rising to 40 and 50 a minute. He gradually became more emaciated and weaker. There were at no time any hæmorrhages from the bowel or lungs. In February both eyes showed optic neuritis with some choroidal atrophy but no hæmorrhages. At this time the corpuscular richness of the blood was—red ·44 of the normal, and the ratio of the white corpuscles to the red 8—600, or 8 times the normal. His dyspnœa gradually became worse and he died. He latterly suffered from much pain in the splenic region. His temperature was febrile throughout, of a hectic type, with an evening rise to about 102°.

He was treated with quinine, arsenic, and iron, but no drugs appeared to have the least influence on the progress of his disease.

Post-mortem made 51 hours after death.

Body much emaciated; very pallid; œdema of lower extremities and scrotum. Abdomen much distended, partly from gas in intestines and partly from dropsy. All the viscera except the spleen are very pallid, also the muscles. Lymphatic glands of neck, mediastinum, and abdomen are much enlarged, of a pale yellowish-white colour, and firmish on section. They are largest in thorax, where they surround the trachea and the larger bronchi; along the latter they advance into the substance of the lungs and form large bosses, chiefly about the centre. The largest are in the left lung. The right middle lobe is pale

and consolidated, and on incision the bronchi are found to be enormously dilated. All the bronchi are full of thick glairy yellow pus.

Heart 5½ ozs. Both ventricles much dilated. Endocardium smooth and shiny. White patch, size of threepenny piece, on apex of left ventricle. Liver 39 ozs. Left lobe adherent to spleen. Consistence firm. Colour reddish, mottled with myriads of minute white points. Kidneys 4¼ and 4¾ ozs., very pale, firm. Relation of cortex to pyramids normal. Walls of gastro-intestinal tract very thin and pale, no enlargement of the solitary or agminate gland tissue (if anything the amount is rather less than normal). Towards the pyloric end of stomach, the mucosa is uneven from the presence of numerous closely adjacent nodules; these are yellowish-white in colour, are situate at the base of the mucosa, and not larger than a pin's head. Brunner's glands are decidedly large.

The spleen is much enlarged, but retains its shape. Capsule thickened; consistence firm; on section the colour is dark purple mottled with numerous patches of a yellow colour. These are of irregular shape, and vary in size from a pin's head to a small bean. In consistence they are quite firm. The spleen weighs 71 ozs. Its greatest length is 10½ inches, and its breadth 7 inches.

Brain 44 ozs.; very pallid; excess of fluid; otherwise nil.

Microscopical examination.—Sections of the spleen are characterised by the presence of large excess of the fibrous elements of the parenchyma. The sections may also be described as an aggregation of areas, each of which consists of three zones. In the centre is seen the malpighian artery, surrounded by large lymphoid cells; external to this is a zone chiefly marked by the presence of fibrous tissue and the absence of the cell element. In the outermost zone there is still a reticulum of fibrous tissue, but its meshes are crowded with red corpuscles. The stained sections show this arrangement quite well, the centre being blue, the middle buff-coloured, and the outer zone red.

The liver merely shows numerous small areas where the hepatic cells are degenerate; these areas are probably the minute whitish points seen at the post-mortem examination. The lymphatic glands are in the condition of advanced hyperplasia. There is no evidence of degeneration-foci in them.

Remarks on the cases of Leucocythæmia and Lymphadenoma.

The two foregoing cases illustrate the often recurring difficulty of putting a satisfactorily correct name to a disease.

In this instance the difficulty arises from the fact that the relations of leukhæmia to Hodgkin's disease are by no means settled or well defined. It is usual to designate cases wherein the white blood corpuscles are vastly in excess, and where the spleen is greatly enlarged, as leucocythæmia. Where, however, the enlargement of the lymphatic glands takes the prominent position, and where the white cells are not markedly in excess, and where the spleen, though increased in size, does not exceed, say, 2 lbs. in weight, then the clinical term for this aggregation is lymphadenoma, and sometimes Hodgkin's disease.

About calling the first case leucocythæmia there is no difficulty. There was a history of malaria. There was extensive enlargement of the spleen and also of the liver, but there was no evidence that the lymphatic glands were increased in size. There was epistaxis and occasionally albuminuria. The white corpuscles were enormously in excess of the normal.

But what should be the exact name for the second case? In this instance there was no direct evidence at the time of the child's admission that the lymphatic glands were enlarged, while the spleen was considerably increased in size. Examination of the blood showed that the red disks were diminished in quantity, while the white corpuscles were somewhat increased. After the lapse of a few weeks the mediastinal lymphatic glands were found to be enlarged, and this enlargement went on increasing with extreme rapidity. There was hectic pyrexia throughout.

In the post-mortem report this case is classed as Hodgkin's disease, and no doubt this classification is on the whole justified. But there were several exceptional circumstances which ought to be taken into consideration. The illness begins with a distinct rigor. On admission to the hospital the spleen is very large, but no signs of enlarged lymphatic glands were detected. The subsequent course of events rendered it more than probable that their increase in size was a later development. In the typical Hodgkin's disease the spleen, though enlarged, is not enormous. In this instance it weighed over 70 ounces, but was the seat of the yellow areas which have been compared to

lumps of suet, and on the presence of which so much stress is laid. There was constant fever, hectic in type.

On the whole, therefore, we are inclined to agree that this case should be called one of Hodgkin's disease, but one wherein the lymphatic glands became affected subsequently to the spleen.

In both cases the microscopical appearances of the liver and spleen were totally different, and though we have attempted to give an account of these appearances, we are well aware that verbal descriptions of microscopical objects are invariably inadequate, and almost always failures.

However, the microscopical examination does one thing; it helps to confirm the suspicion that there is a great difference between the two kinds of cases, and also suggests the doubt whether the "lymphoid" cells are in reality leucocytes.

CASE 3. *Stricture of the Œsophagus.**—Alice H—, æt. 28, married, one child living, two miscarriages, twelve and five months ago, a charwoman by occupation, was admitted under Dr. Donkin into Tillard Ward, on April 30th, 1890, with great difficulty in swallowing her food, either liquid or solid, and having occasional attacks of severe suffocative dyspnœa.

She came originally as an out-patient to the throat department at Christmas time, 1889. She had been in good health up to the beginning of December, 1889, when she began to suffer from slight cough and sore throat. The latter symptom became rapidly worse, and by the time she came to the hospital she found considerable difficulty in swallowing. She was examined with the laryngoscope and nothing abnormal was seen; the cords were perfect, and there was no swelling or obstruction visible in the pharynx. On attempting to pass a bougie, at the moment that its point reached the pharynx, she seized hold of it and dragged it out, saying that it hurt her. Her appearance being that of a somewhat hysterical woman, the obstruction was thought to be due to a spasmodic stricture of the œsophagus. Shortly afterwards she was admitted under Dr. Allchin. Nothing fresh was noticed in her condition. She was emaciated, and stated that she had been losing flesh for some time. She could swallow bread, but with an effort; milk she could take

* Post-mortem Book, vol. vi. p. 19.

fairly well. An attempt was again made to pass a bougie, but with the same result. Nothing fresh was found on laryngoscopic examination. She remained 24 days in hospital, during which time she was two or three times sick, but her swallowing improved somewhat; she gained 10 ozs. in weight.

When admitted in April, she was obviously extremely ill. Her face was drawn and haggard, and her body emaciated. She stated that she had gradually got worse, and now could not swallow at all. She had latterly had several attacks of dyspnœa, on account of which she had gone to the Throat Hospital, Golden Square, where she had seen Mr. Brackenbury, the house physician, who, being unable to take her in there, had sent her on to Westminster Hospital.

No abnormal physical signs were found in the chest or abdomen.

On laryngoscopic examination the vocal cords were healthy in colour and appearance, but they were seen to be fixed in a position of partial adduction, leaving only a narrow chink about ⅛ inch between them. From this position they moved neither on phonation or deep inspiration. The posterior wall of the pharynx appeared to bulge somewhat over the arytenoid cartilages. By leaning over the edge of the bed she was able to swallow a little milk, sucking it up through a tube. She was placed on large doses of iodide of potassium, on the assumption that the œsophageal obstruction might be syphilitic in nature, and fed by nutritive enemata. It was intended to do gastrostomy in a few days, but she rapidly got worse and died on May 5th.

Post-mortem examination made 30 hours after death. Body much emaciated. Fulness of the thyroid region and slight enlargement of the cervical and submaxillary glands. Viscera pallid and exsanguine.

The lower pharynx, œsophagus, trachea, and stomach were removed *en masse*. At the commencement of the œsophagus is a tough malignant stricture which just admits the passage of a No. 12 catheter. Below the point of stricture, which lies at the upper part of the œsophagus, the œsophageal coats are normal; while above it, for a distance of about 1 inch, they are much thickened and of almost cartilaginous hardness. Co-extensive with the induration is a dilatation, that is, the

infiltrated parts are hollowed out. The surface of the dilatation is eroded and pitted from superficial ulceration of the mucosa. This ulceration reaches about as far as the base of the arytenoids. The mucosa and sub-mucosa of the arytenoid region are much swollen from œdema. The growth has spread outwards and forwards so as to involve the lobes of the thyroid body, binding these firmly down to the trachea, and at the same time eating into their substance. In the right lobe the new growth appears at the surface about the centre as a band of dense white fibrous tissue. At the apex of the left lobe is a colloid cyst about the size of a small spanish nut. The aryepiglottic mucosa is swollen and œdematous. In front the growth is considerable enough to cause a distinct bulging inwards of the posterior surface of the trachea. In three lymphatic glands are secondary deposits. In two it is white and soft, in the third, taken from just above aortic arch, it is tough and fibrous.

In the liver two small nodules, not bigger than large pins-heads, were taken from the upper surface. In the lung a nodule about the size of a small pea was found.

The stomach was much contracted and almost fœtal in shape.

The uterus is in the condition of left latero-version, and right latero-flexion. The right ligaments are twice as long as the left. There is superficial ulceration of the os uteri. The uterine mucosa is covered with a red mucoid secretion.

The ovaries are large, flabby, and contain a few small cysts.

The heart, liver, spleen, and kidneys not noteworthy.

Microscopical examination.—Sections of œsophagus show a large number of the so-called nests imbedded in inflammatory and granulation tissue. The original growth is distinctly an epithelioma, while the appearances of the secondary growths in the lungs are those of a lympho-sarcoma, and in the liver those of a fibro-adenoma. This difference between the primary growth and the secondary deposits is worthy of notice since, as a general rule, there is a distinct resemblance between parent and offspring, making allowance, of course, for the position of the secondary growth. Here, however, there is a distinct difference in type between the original growth in the œsophagus and the secondary growths in the lungs and liver.

Remarks.—This case was a very striking one to any person who observed it in its several stages, and once more reminds one

of the extreme difficulty that sometimes occurs in making a diagnosis between hysteria and organic disease. Her age, sex, and general appearance when she was first under treatment, her action in clutching hold of the bougie when an attempt was made to pass it (so like the action of an hysterical patient under similar circumstances), and finally, her improvement in the power of swallowing and slight gain in weight, made it seem extremely unlikely that it was a case of organic disease of the œsophagus.

On her readmission, however, the complexion of the case was completely altered, and it was obvious, at first sight, that she was suffering from a mortal disease.

Three Cases of Aneurysm of the Aorta.

Case 4. (*a*) *Showing result of treatment by rest.**—Thomas W—, an engineer's fitter, æt. 49, was admitted into King William Ward, under Dr. Sturges, in August, 1886. He had always had good health until the beginning of the year 1886. One day he was lifting a heavy iron bar when he suddenly felt something give way in his chest. He was, however, able to continue his work afterwards.

Some few weeks after he noticed a sensation of heaving in the right side of his chest. Since then his breath had gradually been getting shorter. About three weeks before admission he suffered a good deal of pain in the right side of his chest.

He was a moderate drinker. At the age of 19 he had a chancre and a bubo following, but no skin eruption. In 1874 he had right facial paralysis, which gradually almost entirely passed off, but was still slightly apparent on admission. He had otherwise had perfectly good health. His family history was also good.

When admitted he was a well-built, muscular man about 5 feet 10 inches high. There was well-marked heaving pulsation visible and easily felt over an area of about two square inches in the second right interspace close to the edge of the sternum. Over this area, and from the clavicle to the lower edge of the third costal cartilage, there was dulness on percussion. There was also an area of slight dulness between the sixth and tenth ribs on the right side, close to the spine. The breath sounds over this area were feeble. The heart's apex was in the fifth inter-

* Post-mortem Book, vol. vi. p. 48.

space, ½ inch outside the nipple. The heart sounds were clear, the aortic second sound accentuated.

There was no murmur over the aneurysm. The pupils were equal, the left pulse fuller than the right. There was no albumen in the urine.

He suffered a good deal of pain in the region of the aneurysm, and was unable to lie on his right side; but he remained in much the same condition for some months after admission. He was kept in bed, being made to lie quiet, but no special precautions were taken as to his lying absolutely still, flat on his back. He was given rather low diet, but this was not carried to any great extent. He also took 45 grains of potass. iod. daily until October, 1887. Ice-bags were kept over the pulsating swelling for some time, in fact off and on throughout the first year, whenever there was any pain.

In the beginning of 1887 he was placed on the incurable list, and since then he remained in the hospital constantly, except that on several occasions, when the hospital was being cleaned, he went to his home for a week or two.

In February, 1888, after reaching out of bed to take hold of a book on his locker, the swelling suddenly bulged forward and became superficial to the chest wall to the extent of about half a tangerine orange. This was situated in the second right interspace, close to the sternum. It was thought at the time that this swelling would very likely give way and the aneurysm burst, but the violence of the pulsation gradually subsided, and some consolidation of the sac was supposed to have taken place. In March, 1888, after remaining in bed most of the time since admission, he was allowed to get up and go about. He went out for walks and drives, and worked at wood carving.

From time to time he had a little pain in the chest, but never much. The physical signs remained much the same. On December 1st, 1887, a diastolic murmur was plainly heard over the tumour, but afterwards became less evident, and was often entirely lost. In 1889 his larynx was examined, but there was no paralysis of the left cord. He had no difficulty in swallowing. He was about the ward in his usual health on June 3rd, 1890, when he complained of a little pain in the chest; this continued during the 4th and 5th, but he was up and about, and went out for a walk on the 5th. In the evening of this day

he was laughing heartily with some of the other patients at 8.30; when the sister come round at 8.45 he was moribund, and died in a few minutes.

Post-mortem examination made twenty-nine hours after death.

Body well nourished. Rigor mortis well marked. No special external marks. On removing the sternum it was found that the sac of an aneurysm was adherent to the chest wall at the second right interspace, close to the sternum, but the bone was unaffected. The parts from fauces to diaphragm removed *en masse*. Some few adhesions (old) at both apices. Lungs emphysematous and much pigmented. The right upper lobe is the seat of numerous patches of consolidation, which vary in size from a bean to large pin's head. In the larger of these the central parts are caseo-calcareous, the external forming a fibrous capsule. The smaller ones are almost all fibrous tissue. The presence of these indurations has produced a good deal of puckering of the right upper lobe. There are no vomicæ. Besides the fibrous masses alluded to, there are distributed throughout both lungs, right down to the bases, fibrous nodules from which radiate outwards fibrous processes inosculating with the pulmonary tissue; these are quite black from pigmentation. They are probably due to the same cause as the caseo-fibrous masses.

The pericardium is much distended, partly with blood and partly with black clot, in all amounting to nearly two pints. Just below the pericardial reflection, at the front of ascending aorta, is a ragged rent in aortic wall about ¾ inch long.

The heart is, on the whole, not enlarged, although the right ventricle is hypertrophied. There is much subepicardial adipose tissue. The left ventricle does not show either dilatation or hypertrophy, although the aortic ring measures 4¼ inches in circumference. There is no chronic valvulitis, but the corpora arantii are large, and there is some fenestration of the cusps. From about one inch above aortic ring extends an aneurysmal dilatation as far onwards as the last fourth of the transverse arch. The dilatation is chiefly directed upwards, outwards, and to the right.

Immediately above the pericardial reflection is a secondary dilatation from the foregoing; this abuts against and is adherent

to the second right interspace. The aneurysm is about equal in size to a cocoa-nut; it measures 6 inches vertically, and 5½ inches transversely. The secondary aneurysm is about the size of a walnut. Throughout its extent, the aorta (thoracic and abdominal) is in the condition of advanced atheroma, chiefly caseo-calcareous, but there is no trace of thickening anywhere; on the contrary, the walls of the aneurysm are everywhere very thin, especially at the secondary dilatation. The internal surface is extremely uneven, and in places ulcerated. This is the case at the rupture which marks the commencement of a band of ulceration stretching backwards for about 3 inches. Within the aneurysm is only a small quantity of quite recent clot; there is no laminated fibrin.*

The trachea is somewhat flattened by the tumour, so that its sides are approximated. The left recurrent laryngeal appears to be lost on the arch as it turns underneath. The lungs show no signs of pressure. The radial arteries seem normal, but the posterior tibials show patches of chronic endarteritis. Liver 62½ ozs., normal. Spleen 11 ozs., firmer than normal.

Left kidney 6 ozs. Central third of cortex has almost disappeared, the surface here being deeply pitted. The condition is that of simple atrophy of the cortex, there being no trace of embolism, of cicatrisation, or of inflammation. Right kidney 8½ ozs. In both there are several cysts with clear contents. Consistence firm; colour red, and, except at the part alluded to, the surface is quite smooth.

Gastro-intestinal mucosa normal.

Remarks.—This case illustrates the difficulty that so often occurs in estimating the effect of various methods of treatment in cases of aneurysm. It was supposed from the first we think to be a case of a sacculated aneurysm, owing partly to the localised nature of the swelling and the small area of dulness, and any doubt that might have been felt about the matter was set at rest by the sudden occurrence of the localised bulging, the contents of which afterwards apparently became consolidated. Therefore when the man, after having been kept long at rest on tolerably low diet and iodide of potassium, improved so much that he was able to get up and do light work, it was naturally

* For the microscopical appearances of aortitis see Westminster Hospital Reports, vol. ii., 1886, p. 75.

thought that some clotting had taken place in the sac. He was shown by Dr. de Havilland Hall, by permission of Dr. Sturges, at the Medical Society, in the discussion that took place on aneurysm in the early part of 1890, as illustrating the beneficial effect of the treatment to which he had been subjected. What, then, do we find post-mortem? No sacculated aneurysm, but an uniformly dilated aortic arch, with absolutely no sign of a particle of laminated clot. So that obviously the treatment, although doubtless extremely beneficial in prolonging the patient's life, had not produced in the least the effect it was supposed to have done. Of how many other cases of so-called consolidation of an aneurysmal sac is this true? If this case had only been under observation for the few months which a hospital patient usually spends under the care of one physician, no one would have had any hesitation in saying that the improvement was due to clotting having taken place.

Case 5. (*b*) *Producing Paraplegia.**—James A—, æt. 45, an engineer's fitter by trade, was admitted into Bouverie Ward under Dr. Allchin, in April, 1889, complaining of pain in the lumbar region. He stated that he had suffered from pains in the back and limbs for ten years, up to which time he had had perfectly good health, but that during the last twelve months the pain in the back had become very severe and paroxysmal, causing him to bend over forwards during the attacks.

Nothing abnormal was found in the chest or abdomen, and the diagnosis made was that of lumbago. He soon improved, and went out in a few days.

He was readmitted into Burdett Ward on May 5th, 1890, under Dr. Donkin. He stated that he had kept fairly free from pain until January 6th, 1890, since when he had been unable to work owing to the pain and weakness of the legs, which had been so great as to confine him to bed for the last six weeks. He was, however, able to walk to the hospital with the aid of sticks.

On admission he complained of pain in the hips and knees, and weakness of the legs, and said that he had wasted a good deal lately, more especially about the legs. On examining him, there was found to be considerable loss of power in the legs,

* Post-mortem Book, vol. vi. p. 24.

which were wasted. He could hardly move them about in bed at all. The gait was not ataxic, merely weak and dragging. The knee-jerks were absent. The sensation on the outer part of each thigh was impaired but not abolished, elsewhere normal. There was no special loss of power in the arms, although they shared in the general wasting. The electrical reactions of the nerves and muscles of arms and legs were normal. Nothing abnormal was felt in abdomen, but the pressure was chiefly made in the pelvic region. The heart's apex was felt in the nipple line and sixth interspace; the beat was heaving in character. First sound loud and prolonged, second sound clear. No abnormal physical signs in the lungs. There was some prominence of the spines of the last or dorsal and first two lumbar vertebræ noticed, but there was no pain or tenderness on pressure over them. A systolic murmur was heard over this region.

His condition remained much the same for the next few days. He suffered a good deal of pain, but sat up in bed and moved about without making it much worse. His legs became more numb. His bowels were very constipated. His temperature varied between 99° and 101°.

On May 10th he died suddenly, after getting out of bed in the middle of the night.

Post-mortem examination made thirty-eight hours after death. Body emaciated. Projection of spinous processes of vertebræ in lower dorsal and upper lumbar regions; the projection is flat and not angular.

On opening the thorax, the left pleural sac is found to be full of blood and clot, in all amounting to about three pints and a half.

On either side of the vertebræ project into the thorax two soft fluctuating tumours, each the size of a large orange. (These were afterwards found to be merely parts of a large aneurysm compressed by the diaphragm over the column, so as to give the appearance of a double tumour.) About the centre of the left tumour is a ruptured aperture with ragged edges. The diaphragm is stretched over the upper surface of the aneurysm, and is adherent thereto. On removal of the abdominal viscera the aneurysm is found as a fluctuating tumour lying below the diaphragm and behind the liver and stomach. The lower part

of the spinal column and the aneurysm were then removed *en masse*. The aneurysm was then found to be a globose tumour with a diameter of about 7 inches. It springs from the commencement of the abdominal aorta, its aperture lying between the diaphragm and the cæliac axis. The aperture into the sac is oval, and measures 2½ inches in length and 1½ inches in breadth.

Posteriorly the sac is adherent to the vertebral column in the dorsal and upper lumbar regions; indeed, the sac wall is partly formed posteriorly by the necrosed bodies of the vertebræ. These are the eleventh and twelfth dorsal and first and second lumbar. These bodies are in great measure destroyed, so that the aneurysm projects into the spinal canal, where it has pressed upon the last inch of the cord and the corda equina.

Heart 11 ozs., valves competent. Some small but firm vegetations in aortic cusps. Mitral and aortic valves both contain patches of thickening and induration. The aorta is throughout the seat of numerous patches of atheroma.

The left lung is somewhat diminished in size, from the pressure of the blood in the pleural sac. Over against the spot where the aneurysm has burst through the diaphragm, about the centre of the diaphragmatic pleura, is a patch of recent pleuritis. In other respects the lungs and pleuræ are not noteworthy. Liver 68 ozs., normal. Spleen 6 ozs., normal. Kidneys 9 and 10½ ozs., normal. In the right is a cyst about the size of a small walnut; it contains a clear watery fluid.

Remarks.—This case is remarkable in several ways, perhaps most so from the fact that so large an aneurysm should exist with so few symptoms, notwithstanding that it had produced such extensive destruction of the bodies of four vertebræ. When one saw the actual state of the man's spinal column on the post-mortem table, it was impossible to help wondering that he was able to sit up at all, much more to walk about fairly well. After death the cord did not appear to be much compressed by the sac, but probably the state of affairs was very different when the sac was distended during life.

The diagnosis first made was that of peripheral neuritis, of the nature of a double sciatica; but when the electrical reactions were found to be normal, it was seen that the paralysis must be due to some other cause, and pressure either on the nerves of

the sacral plexus or on the cord itself was thought probable. However, examination of the abdomen showed nothing abnormal; no tumour could be felt in the pelvis, and nothing abnormal was noticed except the prominence of the spines of the lumbar vertebræ. No doubt the reason pulsation was not more plainly felt in the umbilical region was that the aneurysm tended to press backwards much more than forwards. Why that should be the case it is difficult to see, as the direction taken by it was certainly not that of least resistance.

Unfortunately, before Dr. Donkin could come to a more definite conclusion as to the cause of the paraplegia, the man died.

The number of published cases of paraplegia produced by pressure of an aneurysm is exceedingly small, and it is noticeable that in a large proportion of them the cause of the paraplegia was not discovered until after death.

We have found the following references to cases previously recorded.

In the Bull. Soc. Anat. de Paris, 1841, p. 95, Renauldin describes a case of "*Aneurysme de l'aorte pectorale descendante avec paraplegie; compression de la moelle.*"

In the Bull. Acad. de Med., Paris, 1853—4, p. 517, Andral records a case of "*Paraplegie par aneurysme aortique,*" in which the patient suffered from pains and weakness in the legs; aneurysm was not discovered till the post-mortem, when there was found an aneurysmal sac eroding the vertebræ in the dorsal region with a diverticulum pressing on the cord and compressing it. A case is recorded in the *Lancet*, 1852, vol. i. p. 374, of an aneurysm "connected with pain in the inferior extremity supposed to be rheumatic," with an account of the autopsy. Frarier, in the Bull. Soc. Anat. de Paris, 1866, vol. xli. p. 47, records a case of "*Aneurysme avec ouverture dans le canal rachidien; paraplegie,*" in which a patient having an obvious thoracic aneurysm was admitted with weakness of the legs, which quickly became completely paralysed. There was found post-mortem erosion of the spine in the upper dorsal region by a diverticulum of the sac which projected into the cavity of the canal.

H. Leach, in the *Med. Times and Gaz.*, 1868, vol. ii. p. 700 records a case of aneurysm opposite the sixth dorsal vertebra

causing erosion of the bone, and compression and flattening of the cord. Symptoms came on about five weeks after admission; there was gradual weakness of the legs, then complete paraplegia, and at the last gangrene of the feet.

J. Coats, in the *Glasgow Med. Jour.*, 1871—72, p. 207, a case in which paraplegia came on gradually, the cause of which was afterwards found to be an aneurysm in the mid-dorsal region, which had eaten through one of the vertebral bodies and caused hæmorrhage into the canal. This was succeeded by suppurative meningitis. The aneurysm was unsuspected during life.

In the Guy's Hosp. Reports, 1871, vol. xvi. p. 389, Dr. Habershon records a case of paraplegia "simulating that due to spinal disease, aneurysmal sac behind the artery." This case is referred to by Dr. Fagge in his Text-book of Medicine, in which he also says "paraplegia from aneurysm is rare."

Further cases will be found recorded in *The Medical Record*, 1876, vol. i. p. 443; *Lancet*, 1881, vol. i. p. 581.

From this it will be seen that paraplegia from pressure of an aneurysmal sac is by no means a common affection, and that the cause, therefore, is not at all unlikely to be overlooked.

Case 6. (*c*) *Treatment by Moore's Operation.**—Henry G—, æt. 46, a bargee, was admitted under Dr. Donkin into Burdett Ward on March 29th, 1890. He had been a bargee all his life, and was in good health up to August, 1889. He had never had syphilis. He drank a good deal of beer. In August he began to suffer from pain in the left shoulder and side of the chest; in December the pain got a good deal worse, and he had to give up work; soon after cough and shortness of breath came on. In the middle of February, 1890, he noticed pulsation in the back, and shortly afterwards a swelling which had gradually been increasing in size. Since August he lost 1½ stone in weight.

On admission: a powerful-looking man, whose face wore an expression of constant pain. Even through his clothes there could be seen a large pulsating swelling in the left interscapular region. On his being stripped, this was seen to be rounded, projecting about 2 inches above the chest wall, and occupying an oval area measuring about 6 inches vertically and 5 inches horizontally. There was marked distensile pulsation of this

* Post-mortem Book, vol. vi. p. 28.

swelling. The left scapula was pushed outwards away from the chest, bringing the point of the shoulder further forward than the right. There was a systolic bruit heard over the centre of the tumour, increased by pressure, and possibly also a faint diastolic murmur. It was impossible to make out whether the ribs beneath the aneurysm were intact or not; no crepitus could be felt.

The pupils were equal and acted to light and accommodation; the right pulse was somewhat fuller than the left. There was no laryngeal paralysis, and no difficulty in swallowing. The heart's apex was in nipple line in the sixth interspace; sounds clear. The physical signs over the lungs were normal, except that the breathing at the left base was feebler than the right. There was no evidence of the pressure of an aneurysm from examination of the front of the chest.

For five weeks no change took place in his condition except that the aneurysm gradually increased somewhat in size, especially in an outward and downward direction. He continually suffered considerable pain. Two casts were taken of his back, which showed the increased size of the tumour. As the aneurysm at the back seemed to be a consecutive one, with possibly no very definite sac, and as it seemed to be gradually getting larger, it was determined to introduce silver wire into the sac in the hope of inducing coagulation. On May 7th Mr. Macnamara did so, passing one yard of silver wire into the sac through a curved tubular needle. The wire passed in without much difficulty up to this point, but then caught and was obliged to be cut off. As there still remained a yard of the amount that it was intended to use, the needle was again introduced, but this time it broke off before any wire was passed in. Next day, however, the operation was repeated, but on this occasion Mr. Macnamara only succeeded in passing in 2 inches.

He went on well till May 10th, when his temperature began to rise, reaching 103·2° on 11th. His back became more painful, and the surface of the tumour became tense and œdematous, the swelling extending upwards chiefly. He suffered a good deal of pain. He took food badly, became rapidly weaker, and died on May 13th.

Post-mortem examination made thirteen hours after death. Body fairly well nourished. Rigor mortis well marked. About

the centre of the posterior aspect of left thorax is a large bulging which pushes forwards the scapula forming a distinct hump. The front of thorax having been removed, it is found that the left chest contains several pints of dark yellow serous fluid. From the beginning of the descending aorta proceeds an aneurysm which extends backwards and outwards behind the left lung and pleura towards the axilla. Viewed from the front it forms a large ovoid swelling at the back of the left thorax. The aneurysm also extends across the spinal column, and appears as a tumour in the right thorax. Here it is about the size of an orange but flatter in shape. In front of this right division descends the œsophagus, and in front of the left lies the completely collapsed lung. In the right pleural sac is excess of fluid. The right lung is emphysematous, but in other respects fairly healthy. The thoracic tumour and the parts to which it was adherent, viz., the spinal column and some of the left ribs, were removed *en masse*. It was found on reflecting the skin of the back that the aneurysm had burst at its outermost part beneath the scapula, the muscles and tissues in this region being sodden with extravasated blood. The aorta was cut up the front and found to be in an advanced condition of atheroma throughout. At the commencement of the descending aorta is the opening into the sac. It is oval, and is about 3 inches long by about 2 inches broad. Posteriorly the wall of the aneurysm where it projects into the back is formed of fibrous tissue, and is for the most part quite thin. The sac is found to contain much clot, that in the right portion being firm and laminated, while that in the left part is black and soft. Within the sac were found two pieces of thin wire, in all about 3 feet long. Within the sac can also be felt the necrosed third, fourth, fifth, and sixth ribs. The third and fourth are quite loose. The bodies of their vertebræ are bare, necrosed, and deeply eroded. The erosion has penetrated into the spinal canal, the dura being thickened and adherent for a length of about 1 inch; its arachnoid surface is quite red from injected blood-vessels. The shape of the aneurysm is due to the spinal column, by which it is apparently divided into two distinct halves. Although not really bilobed, the influence of the vertebral column as a septum has been sufficient to determine a difference in the quality of the clotting. There is no pressure on the left bronchus.

Heart weighs 14½ ozs., valves quite competent. No chronic valvulitis, but on the aortic cusps are festoons of recent vegetations.

Liver 60 ozs., normal. Spleen 5¼ ozs.; capsule thickened and adherent, otherwise normal. Kidneys 5¼ and 5½ ozs., normal. Gastric mucosa normal.

Remarks.—In vol. 70 of the Trans. Roy. Med. Chir. Soc. a case of thoracic aneurysm treated by wiring is recorded by Mr. A. Pearce Gould and Dr. White. In this paper a list of sixteen recorded cases in which the introduction of a foreign body into the sac has been resorted to is given. There was recovery in two cases only. The various substances used have been horse-hair, catgut, watch spring, silvered copper wire, iron and steel wire. The amount of wire introduced seems to have varied greatly, from 1 foot to 26 yards. The operation was first performed by Mr. Moore on a patient of Dr. Murchison's in the Middlesex Hospital (which seems ever since to have been the home of such operations in this country). Moore's case will be found recorded in the Trans. Roy. Med. Clin. Soc., 1864, p. 136. He introduced 26 yards. Death ensued in a few days from pericarditis and suppurative nephritis. This probably resulted from the use of wire which had not been rendered aseptic. A reference of the three last cases may not be uninteresting.

In vol. 69 Med. Chir. Soc. Trans. is recorded a case by Mr. Hulke and Dr. Cayley, in which 40 feet of steel wire were introduced into a thoracic aneurysm in very small coils. This was effectual in producing consolidation, but the aneurysm increased in another direction, the operation was repeated, and death finally resulted three months after the first operation from pressure on the trachea. In vol. 70 of the same Trans. Dr. Pringle records a case under him in which an aneurysm of the upper part of the abdominal aorta was treated by performing laparotomy, and introducing 1 foot (more could not be introduced) of steel wire. There was much difficulty in the operation. Some clotting apparently resulted, and the patient was relieved of some of his pressure symptoms, but he died of exhaustion on the 5th day.

In Mr. Gould's case, the aneurysm was a large one in the right mammary region, partly inside the chest wall and partly

outside. It was rapidly increasing. Thirty-two feet of steel wire were introduced in two parts. Gangrene ensued in the course of a week, and death occurred from exhaustion. Mr. Gould thinks too much wire was introduced, that 10 feet would have been sufficient. The large amount probably increases the danger of gangrene in aneurysms which are superficial, by producing too rapid consolidation, and consequent strangulation of the tissues. Mr. Gould's case resembles the one now recorded, in the fact that the aneurysm had come through the ribs, and perhaps become diffuse even before the operation.

Mr. Christopher Heath, in his Dictionary of Surgery, says, "The introduction of iron wire or of horsehair into an aneurysm with the view of entangling clot need only be mentioned as having been tried unsuccessfully." This seems, perhaps, rather a severe criticism of an operation which has apparently succeeded twice out of sixteen times (and partially also in Mr. Hulke's case), and which is sometimes apparently almost the only method of treatment available in cases which are rapidly hurrying to a fatal termination. In the case above recorded, it seems very probable that the secondary sac had already ruptured, and the aneurysm already become diffuse at the time when the operation was performed, and therefore, even if coagulation had occurred to a greater extent than it did, little benefit would have accrued, as the hæmorrhage into surrounding tissues would have undoubtedly continued. Silver wire hardly appears to have been so effectual in causing coagulation as iron or steel, and certainly it is very troublesome to the operator, owing to its being so soft and so easily doubling up when it meets with any resistance.

Three Cases of Cerebro-spinal Meningitis.

Case 7. Lydia J—,* æt. 40, a cook, was admitted into Adelaide Ward under Dr. Sturges on March 7th, 1890. She was taken ill, on March 1st, with a "cold on her chest," but she did not apparently become very ill until a day or two before admission. She was seen by a doctor on the day of admission. She was then delirious, refusing to stay in bed. He considered her to be suffering from the effects of drink, and recommended her removal to the country, but on the way to the station she became so ill that she was brought to the hospital.

* Post-mortem Book, vol. v. p. 371.

On admission she seemed very ill; she was drowsy, there was subsultus, sordes on lips, her tongue was dry and brown. Temperature 101°; pulse 154, slightly irregular; respirations 60. There was dulness and distant tubular breathing at the right base. There was a rough sound heard with the systole over the base of the heart, apparently pericardial. She rapidly sank and died twenty-four hours after admission. Temperature rose to 106·4° before death.

Post-mortem examination made forty-six hours after death. Body spare but not ill-nourished; skin of abdomen greenish; numerous livid patches on skin. On opening the thorax the right pleural sac is found to contain quite a pint of pus and purulent lymph, which forms a thick layer on the parietal and visceral surfaces. On removal of lung it is found on incision to present no traces of inflammatory consolidation, but to be carnified from the pressure of the fluid in the sac. The apex, which has escaped the pressure, is very œdematous. The left pleura is quite smooth and shiny. The base of this lung is somewhat friable, but presents no other mark of inflammation. The rest of the lung is fairly crepitant. Heart 9½ ozs., normal. Liver, spleen, and kidneys normal. Numerous petechiæ in gastric mucosa; intestinal mucosa normal. Uterus large; fundus full of soft blackish clot. In left ovary is a recent corpus luteum.

Brain 42 ozs. The pia of brain and cord are in the condition of suppurative meningitis. The superjacent arachnoid is everywhere quite smooth. The parts of the brain chiefly affected are the vertex along the longitudinal fissure, the base and sides over the supramarginal convolutions, and the upper and inferior surfaces of the cerebellum. No otitis.

There is little difference between the amount of pus in the anterior and posterior aspects of the cord; the pus forms a thick and continuous layer all the way down the cord.

Case 8. Annie Elizabeth H—,* æt. 15, a domestic servant, was admitted under Dr. Sturges into the West Extra Ward on July 24th, 1890.

She was stated to have been perfectly well until two days before admission, on July 22nd, when she was seized with

* Post-mortem Book, vol. vi. p. 85.

severe headache, and went to bed. She was sick on the evening of this day, and continued so until 10 A.M. on 24th. She rapidly became drowsy, with some delirium.

On admission at 2 P.M. on July 24th, she was delirious, with retraction of the head. Temperature 104·3°; pulse 92, slightly uneven, becoming markedly irregular in the evening; respiration took on Cheyne Stokes' character just before death. There was no strabismus. Pupils were equal, acted to light. Chest and abdomen showed nothing abnormal. She died the same evening.

Her previous history was very good. A maternal aunt died of phthisis, otherwise her family history was excellent.

Post-mortem examination made fourteen hours after death. Body well nourished. Slight post-mortem lividity of posterior aspect of head and trunk. No marks of injury. Brain 51 ozs. Subarachnoid space of brain and cord is the seat of a considerable collection of pus. The meningitis is most extensive over the vertex and down the posterior surface of cord, the evidence of inflammation at base of brain, and on anterior aspect of cord, being much less marked. Nervous tissue of brain and cord normal to naked eye. No tubercles on pia mater. No otitis media. Bones of skull, &c., normal.

All the thoracic and abdominal viscera are quite healthy except the ovaries, which are cystic.

CASE 9. Frederick D—,* a carman, æt. 19, was admitted into King William Ward under Dr. Sturges on September 25th, 1890. He was in his usual health, which was excellent, on September 17th. He then got wet through and slept that night in his wet clothes. Next day he was not quite well, but continued at his work until the morning of the 22nd, when he shivered, had pains all over him, especially down his back, and severe headache. He was obliged to give up his work and take to his bed. These symptoms continued. He became drowsy, and was sick. On 24th his drowsiness passed into coma, with occasional outbursts of delirium.

On admission he looked extremely ill; he was muttering and picking at the bed-clothes, very difficult to rouse at all, and then gave an unintelligible answer. His face was flushed, eyes being very much suffused, especially the right. His tongue was dry

* Post-mortem Book, vol. vi. p. 135.

and covered with brown fur; his teeth dry and lustreless. Temperature 107°. Pulse 124, slightly irregular. Double external strabismus. Ophthalmoscopic examination showed the fundus and disc to be acutely hyperæmic.

He rapidly sank, and died the same evening.

His previous history was excellent, also the family history. No other cause except the wet was apparent.

The post-mortem examination was made seventeen hours after death. There was nothing noteworthy except the brain and cord. There were no marks of violence.

The inner surface of the skull-cap was very dry. The surface of the brain was covered with a layer of purulent lymph, thickest over the vertex, especially in the region of the sylvian fissures. The lymph occupied chiefly the fissures, but near the median line of the vertex covered the whole convolutions.

The brain weighed 59 ozs. The brain substance appeared normal.

The lateral ventricles were somewhat dilated and contained about 1 oz. of clear fluid.

The inflammation extended all down the cord, the pus being collected especially down the posterior aspect, and laterally.

Both tympanic cavities were healthy, as also was that of the nose.

Remarks.—These cases have been reported as illustrating the extremely rapid course taken by some cases of suppurative meningitis. In one case the cause seemed definitely to be a severe wetting. One case was pyæmic in nature, and secondary to an acute suppurative pleuritis; the exciting cause of the third was quite obscure. The question of epidemic cerebro-spinal meningitis naturally arises, but there was no reason to suppose that these cases come under such a category. They occurred at intervals of some months, came from different localities, and there was no such epidemic reported as then existing in London. In each case it was definitely ascertained that no local cause, such as otitis media, was present.

CASE 10. *Tubercular Tumour of the Third Ventricle.**—Albert M—, æt. 5 years, was admitted into St. Margaret's Ward under Dr. Donkin on April 14th, 1890.

* Post-mortem Book, vol. vi. p. 39.

He had had good health until February, 1889, when he had a fall. Shortly afterwards he was found to have lost power in the right arm, and to a less degree in the right leg. This came on suddenly; three months before admission his eyelids were noticed to droop. On April 12th the child complained of pain in the left ear and headache; his face became flushed, and he became drowsy. There was no convulsion.

On admission the child was in a condition of somnolence passing into coma, and unless disturbed was not restless; when disturbed he became irritable. The pupils were equal and reacted slowly to light; there was external strabismus of both eyes, but more of the left; there was ptosis of both eyelids, but also more marked on the left side; no optic neuritis. There was considerable rigidity of the right arm and leg, but becoming less when the child was roused. The knee jerk and ankle clonus were more marked on the right side than the left. There was no apparent anæsthesia, but sensibility was generally dulled. There was no perforation of the membrana tympani, or discharge from the ear.

For five weeks the child continued in much the same condition. The right arm was always found to be more rigid than the left, but the degree varied. The ptosis and strabismus became greater. If pinched on the face the head was still turned away. He passed his motions and urine in bed, but always swallowed well. The temperature was slightly raised throughout, rising to 100° on most evenings.

The question of operative interference was raised, but owing to the fact that the symptoms rather pointed to a more diffused affection than would be likely to be removable by operation, and the opposition of the parents, the idea was given up. On May 24th he became more deeply comatose, his temperature rose to 104°, and next day to 107·6°, and he died on May 27th.

Post-mortem examination made nineteen hours after death. Body fairly well nourished. Feet in position of extreme extension. Fingers tightly clenched. No marks.

Brain 43 ozs. Very slight flattening of convolutions. No evidence of meningitis. Excess of subarachnoid and intraventricular fluid. The floor of the third ventricle bulges downwards; it has a translucent appearance, and is much stretched from the pressure of the intraventricular fluid.

On cutting through corpus callosum and fornix, there is disclosed a hardish tumour about the size of a spanish nut. It lies in the third ventricle and abuts on both thalami. The tumour, which has a bilobate appearance, contains areas of caseation, and is tubercular in character. The tubercular induration has further invaded the left thalamus, both the crura and the anterior half of the pons, all which structures are practically replaced by tubercular caseations. The adjacent or inner part of the right thalamus is also destroyed. Both lateral ventricles are much distended by fluid, especially their posterior coruna. The wall of the left ventricle and the adjacent white matter are softened and disintegrated. The rest of the cerebrum, the cerebellum, and the spinal cord are to the naked eye quite normal.

Considerable œdema of epiglottis and of aryepiglottic folds. Both lower lobes of the lungs are blue in colour, consolidated and friable (lobular pneumonia). Pleuræ normal. Heart 2¾ ozs., normal. Liver 20 ozs., one or two small yellow tubercles beneath capsule. Spleen 1¾ oz.; a few yellow tubercles about the size of a pin's head. Kidneys 3 to 3½ ozs., *nil.*

In the thorax is one large caseo-cretaceous gland.

The right middle ear is filled with a thick yellow viscid fluid. The ossicles are loose.

The left tympanum is normal.

Microscropical examination.—The cerebral tumour presents the usual characteristics of the tubercular caseations met with in cerebrum or cerebellum.

The spinal cord shows well-marked descending degeneration in one lateral column (the right). In position (*i.e.*, as regards the lateral column) the degeneration area is internal, abutting immediately against the grey matter of the coruna, especially the posterior. It is most extensive in the upper part (cervical enlargement) of the cord, gradually diminishing in amount until in the lumbar region it may be said to have disappeared. There is some evidence that the left lateral tract is also affected.

Remarks.—The chief points of interest in this case seem to be the presence of the tumour in the third ventricle; the palsy with rigidity of the right arm and leg; the double ptosis with external squint; and lastly the sleepiness or torpor. This con-

dition began as drowsiness, passing on through sleepiness and torpor to coma. In connection with a case recorded in the Transactions of the Pathological Society (vol. xxxvii. p. 50), where a similar condition was found to coexist with a gliomatous tumour in the third ventricle, these phenomena seem worthy of special notice.

The palsy of the third nerves was produced by the pressure of the tumour on these nerves. Owing to its size and position, the tumour destroyed or impaired the nerves to the descending tract. This was shown in life by the palsy with rigidity, and after death by the degeneration-sclerosis of the lateral column. Taken in connection with the presence of the tumour in its actual position, the lesion in the lateral column is not surprising, but is worth recording since it is not universal experience that a tubercular mass gives rise to structural alterations in the cord.

CASE 11. *A Case of "Fever" of undetermined Origin.**—Edward R—, æt. 32, occupation unknown, was admitted under Dr. Donkin on July 7th, 1890.

Little is known of his history; he was stated to have been a soldier, and to have had sunstroke in India some years previously. He had been ailing for a few days before admission, but had not been seriously ill until the day before he came in. When admitted he was extremely ill, very delirious, constantly trying to get out of bed. Temperature 104°; tongue dry and brown in centre. There were no physical signs in his chest.

He rapidly became worse and died in a few hours.

Post-mortem examination made fourteen hours after death. Body well nourished. Rigor mortis persistent. Considerable post-mortem lividity of head, neck, and posterior surface of body. No marks of violence. Brain 54 ozs. Beyond a marked fulness of the veins and noticeable puncta cruenta, the brain and cord (membranes and nervous tissue) are to the naked eye quite normal. Larynx *nil.* Mucosa of trachea and bronchi is throughout much reddened from injection of the finer blood-vessels. Both lungs are fuller of blood than usual, but they are perfectly crepitant, and there is no trace of pulmonary inflammation anywhere. The left pleural surfaces are adherent

* Post-mortem Book, vol. vi. p. 68.

at the apex (old), and this sac contains 8 to 10 ozs. of slightly turbid fluid, but the pleural surfaces are, except at apex, quite smooth and shiny. On both sides of chest the subpleural parietal vessels are well marked. Heart, with pericardium, weighs 16 ozs. Pericardium universally and firmly adherent. Endocardium normal. Muscle red and firm.

The mediastinal tissue is much more abundant than usual. This increase is due partly to overgrowth of the fibrous tissue element, partly to enlarged but not caseating lymphatic glands, and partly to the presence of a persistent thymus. The latter, when sectioned, exuded a characteristic, thin milky juice.

Peritoneum and mesenteric glands normal. Liver 65 ozs.; capsule, which strips easily, is adherent to diaphragm; colour yellowish; consistence soft and friable (fatty). Spleen 10 ozs., adherent to adjacent tissues and viscera. In centre of convex surface is a yellow patch about the size of a shilling. It is a calcareous thickening of the capsule. Splenic parenchyma very soft. Kidneys 6 and 6½ ozs. Capsules slightly adherent in places, but on the whole the surface is smooth. In both kidneys are numerous spheroidal yellow bodies resembling miliary tubercles both as to size and general appearance. They are situated chiefly in the cortex. In other respects these organs are not noteworthy.

Bladder and ureters normal.

Gastro-intestinal mucosa quite normal.

Microscopical examination.—Pia mater normal; no sign of inflammation, tubercular or otherwise. Lungs normal.

Kidneys.—The yellow spherules are areas of necrosis, *i.e.*, they are spheroidal patches in the earlier condition of caseation, a stage wherein the structure of the original parts still remains visible, but is almost sharply delimited from the adjacent healthy tissue. The delimitation is well shown by staining reagents, *e.g.*, alkalin methylin blue stains the necrosed areas deeply and distinctly, but the circumjacent healthy tissue scarcely at all. In the centre of all the necrosed areas is a colony of micrococci. The colonies are of fair size, and probably situate in a capillary vessel, but there is no evidence of inflammatory activity; there is no hyperæmia of the adjacent vessels, and leucocytes are conspicuous by their absence. The areas are therefore necrotic patches and are not abscesses or due to inflammatory softening.

This case is a good example of how difficult it often is to say not only what is the disease, but what is the cause of death. Practically the symptoms amounted to pyrexia and delirium, passing into coma. The result of the post-mortem examination failed to throw much light on the matter, though certain positive and negative facts were noted. There was adherent pericardium, showing that there had been severe inflammation at some past epoch, and leaving behind an impaired condition of the heart, a heavy handicap for a subsequent disease under any circumstances. It is quite probable that the condition of the heart accelerated death, but this does not explain the nature of the disease. It was obviously some acute febrile disorder, arguing at least from the symptoms and from the post-mortem appearances of the viscera. Then there were the yellowish round nodules in the kidneys. The resemblance of these bodies to miliary tubercles suggested the notion that it was a case of acute tuberculosis. Had this been so the pia mater would, when examined microscopically, have probably supplied the necessary information. But the pia was as normal microscopically as it was to the naked eye. There was no trace of tubercle there, or indeed elsewhere, or any evidence of any kind of inflammation. For the yellow nodules were found to be not inflammatory, but necrotic.

Taking all the circumstances into consideration, it is probable that it was a case of blood-poisoning of specific origin; some acute infective disorder, in which the necrotic foci in the kidney are to be regarded as secondary or embolic rather than primary or inflammatory.

OFFICIAL REPORTS.

REPORT

OF THE

POST-MORTEM EXAMINATIONS MADE IN 1889.

By Dr. R. G. HEBB.

Cases recorded			199
Males		121	
Females		78	
General Diseases:			
Diphtheria		5	
Measles		1	
Erysipelas		1	
Enteric fever		2	
Rheumatic fever		1	
Pyæmia		1	
Tuberculosis		13	
Congenital syphilis		1	
Delirium tremens		1	
Leukhæmia		1	
		—	27
Diseases of the Nervous System:			
Cerebrospinal meningitis		3	
Chronic hydrocephalus		3	
Arachnoid hæmorrhage		1	
Cerebellar tumour		2	
Convulsions		1	
		—	10
Diseases of the Circulatory System:			
Pericarditis, acute		3	
Endocarditis, acute		1	
,, chronic—			
Mitral	9		
Mitral and tricuspid	2		
Mitral, tricuspid, and aortic	6		
Mitral and aortic	3		
Aortic	3		
Aortic and pulmonary	1		
	—	24	
Congenital		1	
Aneurysm of aorta		1	
Embolism of pulmonary artery		1	
		—	31

Diseases of the Respiratory System:		
Bronchitis, acute	2	
Bronchitis, chronic, including emphysema	8	
Broncho-pneumonia	16	
Pneumonia	13	
Phthisis	11	
Pleuritis, acute	3	
Empyema	1	
	—	54
Diseases of the Digestive System:		
Ulcerative pharyngitis	1	
„ colitis	1	
Ulcer of duodenum	1	
„ vermiform appendix	1	
Perityphlitis	1	
Abscess of liver	1	
Cancer of stomach	4	
„ „ intestine	2	
„ „ bile duct	1	
„ „ pancreas	1	
Diarrhœa with emaciation	10	
Peritonitis	2	
	—	26
Diseases of the Lymphatic System:		
Tuberculosis, chronic	1	
Cancer	3	
	—	4
Diseases of the Urinary System:		
Chronic renal—		
Contracted granular kidney	6	
Large white kidney	3	
	—	9
Diseases of the Generative System:		
Pelvic cellulitis	1	
Cancer of breast	2	
	—	3
Diseases of the Organs of Locomotion:		
Morbus coxæ		3
Injuries and Surgical Operations:		
Revolver wound	1	
Cut throat	1	
Burn	3	
Drowning	2	
Asphyxia	2	
Rupture of liver	1	
Crushed leg	1	
Fracture of thigh	1	
„ skull	1	

INJURIES AND SURGICAL OPERATIONS—*continued.*		
Excision of jaw	1	
Gastrostomy	1	
Hernia	6	
Stricture of Urethra	1	
Extravasation of urine	1	
Amputation of penis	1	
Ulcer of leg	1	
Necrosis of tibia	1	
Ablation of fundus uteri	2	
,, uterine myoma	1	
Ovariotomy	1	
	—	30
UNCLASSIFIED		2

Remarks and short notes on most of the foregoing cases. The Numbers refer to the folios in vol. v. of the P. M. Registers.

Diphtheria.—99. Admitted for tubercular peritonitis, and was much benefited; contracted the diphtheria in hospital. The membrane extended from fauces to bronchi, ending in suppurative bronchitis. Extensive tubercular peritonitis and pleuritis, and a few miliary tubercles in lungs. 110. Respiratory mucosa from nares downwards as far as upper part of trachea covered with membrane. Both lungs are studded all over with spheroidal hæmorrhagic consolidations. Right middle suppurative otitis. 137. Membrane from fauces to larger bronchi, below which the inflammation ends as suppurative bronchitis. Well-marked gastro-enteritis. Rickets. Tracheotomy. 224. See also a case classified as broncho-pneumonia, and compare "ulcerative pharyngitis," 245. 225. Membrane from vocal cords to medium-sized bronchi, below which the inflammation ends as suppurative bronchitis; pneumonia; gastro-enteritis, and enlarged mesenteric glands. Tracheotomy. 271. Suppurative tonsilitis; membrane first appears in larynx, and recommences about tracheal bifurcation, ending as suppurative bronchitis. Pneumonia. Tracheotomy. In this case it was a question whether the child, who was 8 years of age, had not contracted the pneumonia first, and that the diphtheria supervened. Microscopical examination of the lung shows that the capillaries of the alveolar walls are engorged and tortuous, that

the alveoli are crowded with cells and some fibrin. The distribution of the fibrin is very unequal in amount and in location. There is also much difference in the characters of the cells. All the infundibula are crowded with small indifferent cells, while in many of the alveoli the cells are obviously catarrhal in character. Hence the microscopical appearances present a combination of catarrhal and croupous forms of pneumonia, but I am inclined to think from observations on other cases that the pneumonia was diphtheritic, and hence essentially bronchial in origin and catarrhal in nature.

Enteric fever. — 221. Ulceration of ileum and rectum. Patient died after severe arterial hæmorrhage, no doubt from the rectal ulcers. No ulceration of colon.

Rheumatic fever.—196. Case of prolonged hyperpyrexia. Post-mortem appearances negative. Microscopical examination of blood from heart shows large numbers of bacteria of two kinds; one in clusters, the other bacilli forming chains.

Pyæmia.—234. Pus in pleural and peritoneal sacs; numerous abscesses in right kidney, which is about half the size of left. No obvious cause.

Tuberculosis.—117. Patient, aged 2½ years, was admitted for infantile palsy. The palsy of the left leg was complete. But some weeks after admission symptoms of broncho-pneumonia set in, and these were soon followed by the phenomena of tubercular meningitis, of which disease the child died. Besides the tubercles in the pia mater, there were several small caseous nodules in the lungs, but no miliary tubercles were detected therein. There were a few caseous nodules in the mesenteric glands, and these were enlarged. On microscopical examination of the cord, it was found that although the severity of the disease is most marked in the lumbar region, the anterior cornu is affected all down the left side, and this cornu is also diminished in size and altered in shape. In this left cornu the multipolar cells are greatly lessened in number in the cervical region; in the dorsal they almost disappear, and in the lumbar the grey matter is replaced by a thin fibrous tissue traversed by numerous blood-vessels. The left sciatic is distinctly smaller than the right, and sections show a number of withered tubules. In some respects the clinical aspect of the

case and the microscopical appearances are in accord—*i.e.*, the condition of the lumbar region agrees with the phenomena observed during life; but it is noteworthy that while the cord is affected all the way up on the left side, there was no obvious weakness of the parts supplied by the cord above the lumbar region. 182. Tuberculosis of lungs, liver, spleen, kidneys, lymphatic gland, and ulcers of stomach and intestines. 193. Tubercular meningitis; tubercles in lungs, spleen, kidneys, and lymphatic glands, and ulcerations of stomach and intestines. 210. Tubercular caseous nodules in brain; tubercles in lungs, liver, kidneys, and ulceration of stomach, intestines, and bladder. 211. Age 32 years. Tubercular meningitis. Mesenteric glands form a large caseo-cretaceous mass. No recent tubercles anywhere except in brain. 226. Tubercular meningitis; caseous nodules in cerebrum and cerebellum; tubercle in lungs; mesenteric glands large and caseous. 232. Tubercular meningitis. Tubercular phthisis with vomicæ; tubercles in kidneys, and tuberculosis of vesiculæ seminales. 242. Age 49. Tubercular meningitis; tubercles in lungs, liver, kidneys, spleen, and ulcers of intestine. 276. Tubercles in lungs, liver, and kidneys. Lymphatic glands everywhere much enlarged and caseous. 283. Tubercular meningitis; tubercles in lungs, liver, kidneys, and lymphatic glands; ulcers of stomach and intestines. 299. Tubercular meningitis; tubercles in lungs, liver, kidneys, and lymphatic glands. 309. Tubercular meningitis; tuberculosis of lungs and lymphatic glands; ulcers of intestines. 313. Tuberculosis of lungs, liver, kidneys, and spleen. 146. See under lymphatic system a case which might fitly be included in this category.

Leukhæmia.—264. Weight of spleen 99 ozs., of liver 132 ozs.; kidneys granular; heart 17 ozs.; aortic cusps much thickened; calcareous aortitis.

Nervous System.

Cerebro-spinal meningitis.—124. The surface of brain, basal as well as vertical, is covered with pus, the inflammatory exudation spreading down cord. Broncho-pneumonia; double otitis media. Prepuce recently removed surgically. 177. Sur-

face of brain and cord covered with pus. Broncho-pneumonia. Right sterno-clavicular articulation full of pus, and the cartilages eroded. 172. Cerebro-spinal meningitis and abscess after otitis media. Right ovary is represented by a double dermoid cyst. The left ovary is a simple cyst. Myoma uteri.

Chronic hydrocephalus.—124. Ventricles much dilated. Choroid plexus ill developed. Fornix adherent to choroid plexus and optic thalamus. Cerebro-spinal meningitis. Microscopical examination of cord showed that the grey matter contains an extraordinary quantity of blood-vessels and very few cells. 257. Well-marked rickets. Left side of head much larger than right. Ventricles much dilated. Sclerosis of white matter in temporo-sphenoidal lobes. The velum interpositum is much atrophied. 260. Ventricles much dilated; sclerosis of white matter of both frontal lobes; and yellow softening of grey matter of left frontal region and small portion of right frontal region. Broncho-pneumonia.

Arachnoid hæmorrhage. — 103. Large hæmorrhage into intermeningeal cerebral space, with depression of cortex. Hæmorrhage into gastric mucosa. Right latero-version of uterus.

Cerebellar tumour. — 189. Large tubercular caseations in cerebellum. Chronic otitis media. (*See* Westminster Hospital Reports, vol. v. pp. 188—191.) 222. Sarcomatous tumour in cerebellum, with secondary deposits in brain, and along posterior surface of cord. (*See* Westminster Hospital Reports, vol. v. pp. 191—194.)

Circulatory System.

Pericarditis, acute.—105. Pericardial sac full of pus, and surfaces covered with thick, shaggy lymph. Empyema of both sides. Age $1\frac{8}{12}$. 303. Pericardial surfaces covered with shaggy lymph. Both pleuræ are inflamed. 286. Pericardial sac contains about 12 ozs. of dark yellow turbid fluid, and the surfaces are covered with shaggy lymph. Chronic aortic valvulitis. Recent vegetations on aortic and mitral valves. Shape of heart globular. Left ventricle much dilated. Liver cardiac. Lungs much compressed from fluid in pleural sacs.

Endocarditis, acute.—249. Clot weighing 3 ozs. in white matter outside lateral ventricle, and towards posterior part of right hemisphere. Recent vegetations in mitral. Caseous infarcts in kidneys and spleen. This child came to me as out-patient on July 9th, 1889, for chorea and pains in chest (duration, three weeks: cause, fright). At this time the heart sounds were normal, but by September 3rd there was an audible murmur, and she was admitted. The temperature ranged between 98° and 102°, and the anæmia was progressive. On October 3rd was seized with vomiting, soon became unconscious, and died in a few hours.

Endocarditis, chronic.—135. Mitral and aortic cusps much thickened; hyperpyrexia. 160. Mitral stenosis; infarctions of lungs; cardiac liver and kidneys. Congenital malformation of toes. 206. Mitral stenosis; pneumonia; infarcts of spleen; yellow softening of cortex. 250. Mitral stenosis; infarcts in lungs; right ovary cystic; pelvic cellulitis and peritonitis; os uteri stenosed. 265. Mitral stenosis. Right heart much hypertrophied. Diffuse areas of congestion infarcts. Microscopical examination of lung shows that the pulmonary veins are much hypertrophied, resembling the renal arteries in a case of chronic Bright's disease. 270. Pericardium adherent; mitral stenosis. 291. Right heart very greatly hypertrophied. Both auricles in free communication from almost complete absence of septum. Mitral stenosis. 300. Mitral stenosis; lungs cardiac; liver cardiac; infarcts in lungs. 315. Mitral stenosis; left pleura much thickened and adherent. 113. Mitral and tricuspid stenosis. Yellow softening of right ascending frontal and parietal convolutions, the degeneration passing inwards as far as the basal ganglia. 235. Stenosis of mitral and tricuspid. Right pleura much thickened and adherent. 129. Pericarditis; vegetations in tricuspid, mitral, and aortic; right kidney atrophied; left contracting and granular; ovaries large, hard, and cystic. 238. Pericarditis. Chronic endocarditis affecting tricuspid, mitral, and aortic valves. Calcareous atheroma of aorta. Liver almost spherical from chronic capsulitis. Left oblique inguinal hernia. 241. Pericarditis. Chronic endocarditis affecting mitral and aortic valves. Recent vegetations on tricuspid, mitral, tricuspid, and aortic; viscera generally, cardiac. Empyema in right pleural sac. 243. Old and recent

inflammation of aortic, mitral, and tricuspid valves. Besides the valvulitis, the endocardium is also affected by direct inoculation. Aneurysm of the popliteal, which has recently burst. All viscera cardiac. 282. Pericarditis; chronic inflammation of aortic, mitral, and tricuspid valves; viscera cardiac. 287. Chronic inflammation of mitral and aortic valves; recent vegetations on tricuspid, mitral, and aortic; viscera cardiac. 141. Chronic and recent inflammation of aortic and mitral valves; all viscera cardiac. 228. Chronic inflammation of mitral and aortic valves; viscera cardiac. 304. Chronic aortic and mitral valvulitis; cerebral hæmorrhage. 153. Aortic valvulitis; heart dilated and hypertrophied; well-marked aortitis; one aortic cusp fenestrated. During life a loud, musical, diastolic murmur audible all over chest, but loudest in sternal region. Viscera cardiac. 178. Chronic aortic valvulitis; cusps adherent, and one having large perforation at base; viscera cardiac. 194. Chronic aortic valvulitis; heart muscle very pallid. On microscopical examination, osmic acid gave no reaction with the muscle fibres of heart. Transverse striation almost lost. The case was brought in dead. 301. Stenosis of cusps of pulmonary valve; these cusps are thickened and adherent, leaving central circular aperture; the pulmonary artery immediately above is much dilated. The aortic cusps are thickened and recurred. The right ventricle is greatly hypertrophied, its maximum thickness being $\frac{6}{8}$ inch, while that of the left ventricle is only $\frac{4}{8}$ inch. All viscera cardiac.

Congenital cardiac.—284. Interventricular septum defective at base. Pulmonary artery extremely stenosed at orifice. Only two cusps to pulmonary valve. On aortic and tricuspid valves are numerous coarse and hard vegetations. The aorta lies above septum, so that it communicates with both ventricles. This child, aged 5 years, was brought to me as out-patient some months before death for shortness of breath and fits of unconsciousness, from which she had suffered for the past three years; was in condition of extreme cyanosis. Ends of fingers and toes much clubbed. Bronchitis. Loud systolic bruit all over, but most marked over the pulmonary area. Died suddenly at home. Under this heading might also be included cases 291 and 301, both of which are noted in the preceding paragraph.

Aneurysm of aorta.—186. Aneurysm spheroidal in shape, with diameter of 2½ inches, situated just above aortic ring; walls thin; contents decolorised, but not laminated clot. From its size and position it appears to have exerted some pressure on the pulmonary artery. Viscera cardiac.

Embolism of pulmonary artery.—127. Veins of right lower extremity filled with firm clot, which reaches to about one inch above Poupart's ligament. From the right branch of the pulmonary artery was withdrawn a firm decolorised clot about one inch long. It resembles the clot in right iliac vein, and differs altogether from the soft black clot found in the pulmonary vessels and right heart. The patient died almost instantly after an action of the bowels. The veins of the legs are varicose, and the skin scarred and pigmented.

Respiratory System.

Chronic bronchitis.—98. Emphysema; suppurative bronchitis; perihepatitis; chronic pelvic peritonitis, with dropsy of fallopian fimbriæ; ovaries large and cystic. When admitted, was suffering from urgent dyspnœa, which she stated had come on since the removal of tonsils a few days before. At post-mortem examination the wound was found to have quite healed. 136. Emphysema; chronic pleuritis; œdema; congestion, and bronchitis. Right heart enormously hypertrophied. Cavities dilated, and full of soft black clot. Viscera cardiac. 159. Emphysema; chronic pleuritis; œdema, and suppurative bronchitis; hypertrophy and dilatation of right heart; viscera cardiac; pelvic peritonitis; fallopian fimbriæ much dilated.

Broncho-pneumonia.—224. Was admitted just seven weeks before death for diphtheria. Membrane on soft palate, fauces, and tonsils. No physical signs in chest, and no albumen. In a few days albumen was found in urine; and as the membrane disappeared, so signs of pulmonary inflammation set in. This was soon followed by otorrhœa and palsy of palate and ocular muscles. On post-mortem examination, suppurative bronchitis and broncho-pneumonia, involving the whole of the left lung and several areas in the right. Mediastinal glands large and caseous. Peyer's patches swollen and hyperæmic.

Pneumonia. — 106. Grey hepatisation right upper lobe; emphysema. 126. Grey hepatisation, right lower lobe; chronic aortic valvulitis; recent vegetations on mitral. 155. Grey hepatisation right upper lobe; chronic aortic valvulitis. 185. Grey hepatisation right lung; chronic pleuritis; chronic and acute aortic and mitral valvulitis. 187. Red hepatisation of left lower lobe. Patient admitted for eclampsia, and abortion was induced in hospital. There was only a very small quantity of albumen. Microscopical examination of kidney did not reveal any marked structural changes such as are seen in even early conditions of chronic renal. The epithelium of the tubules is swollen and everywhere shows marked tendency to split up into fibrillæ. The basement membrane is everywhere very coarse. 213. Grey hepatisation left lower lobe; acute pleuritis of both sides; pericarditis; recent vegetations on mitral; recent infarct in spleen. 246. Age 7 months. Acute pleuritis of both sides; both lower lobes in condition of red passing to grey hepatisation. 251. Grey hepatisation of right upper lobe; red to grey hepatisation of both lower lobes. 252. Grey hepatisation right lower lobe. 258. Grey hepatisation right lower lobe; left lower lobe in condition of engorgement. Both right and left pleuræ are much inflamed. 266. Grey hepatisation right upper lobe; liver fatty; kidney contracted, granular, cystic. 278. Red hepatisation both lower lobes; phthisis. 307. Grey hepatisation of right lower lobe, red of left lower lobe; right kidney atrophied; left hypertrophied.

Phthisis. — 101. Pleuræ much thickened and adherent; upper lobes much fibrosed and excavated; rest of pulmonary tissue beset with closely-packed tubercles, pigmented and recent. Adherent pericardium; liver amyloid; kidneys large, white; both testicles withered in inguinal canals. 140. Old and recent pleuritis; lungs fibrosed and excavated in upper halves; in lower halves broncho-pneumonic consolidations; tubercles partly caseous and partly fibrous; adherent pericardium; aorta atheromatous; tubercular ulceration of intestines. 188. Pleuræ thick and adherent; lungs in condition of tubercular pneumonic phthisis; numerous small vomicæ; tubercular ulceration of intestines; mucosa of larynx from epiglottis to cords much ulcerated. Uterine mucosa covered with a thin layer of yellow

grumous material; there was no ulceration however, nor did microscopical examination give any evidence of tuberculosis; uterus retroflexed and in condition of subinvolution. 204. Pleuræ thickened and adherent over upper halves; lungs in condition of tubercular pneumonic phthisis. Considerable excavations; on the left side one has perforated the pleura, and the sac contains much fœtid purulent fluid, while the adjacent pulmonary tissue is gangrenous. Tubercular ulceration of intestine. Numerous fistulous passages about rectum, which burrow among the thickened pelvic connective tissue. 205. Pleuræ thickened; recent pleuritis with exudation into left pleural sac; tubercular pneumonic phthisis with excavations; chronic aortic valvulitis. 248. Left chronic pleuritis; right normal; tubercular pneumonic phthisis with a few small excavations; ulcerations in the intestines. This patient, a barman, was admitted for hæmoptysis, and in a short time well-marked phenomena of delirium tremens supervened. This continued for five days. The temperature, always high, rose to 108° just before death.

Acute pleuritis.—259. Respiratory mucosa from fauces to cords red and swollen, the aryepiglottic folds being most affected. In both pleural sacs is a large quantity of lymph and turbid fluid, by which the lungs are much compressed. There is, however, no trace of pneumonia. Excess of fluid (turbid) in pericardial sac, the surfaces of which are covered with a thickened layer of recent lymph. In this case the illness began three days before admission with sore throat. Dyspnœa only set in the day before admission. Death next day. 316. Yellow lymph and purulent fluid in right pleural sac; lung much compressed, but no pneumonia; pericardium covered with shaggy lymph, and sac contains a quantity of turbid fluid. 275. Purulent fluid and yellow lymph in both pleural sacs; no pneumonia; pericardial sac contains a quantity of turbid fluid.

Empyema.—145. Left pleural sac contains many ounces of turbid greenish fluid and air; lung quite collapsed; small hole in pleura communicating with a bronchus. The left chest of this child, aged $1\frac{1}{12}$, had been aspirated the day before death, and 5 ozs. of pus withdrawn. This condition ought also to be termed pneumothorax.

Digestive System.

Ulcerative (sloughing) pharyngitis.—245. Mucosa at back of mouth—*i.e.*, of pharynx, fauces, palate, &c.—is much swollen, and in places ulcerated; abscess outside both tonsils; no membrane, but suppurative inflammation of respiratory mucosa from larynx to bronchi; skin dry and harsh, but no definite peeling. In this case it was difficult to decide during life between scarlet fever and diphtheria. The illness began with swelling of neck and discharge from nares. On admission the cervical glands were enlarged, and there was a bright red rash on body and limbs. No albumen. Shreds of membrane were being constantly coughed up, while the discharge from the nose continued until death. In children it must always be difficult to decide between diphtheria and scarlet fever, when the phenomena and symptoms occur as they did in this case—*i.e.*, membrane on fauces, discharge from nose, with bright red rash. The presence or absence of albumen would go for little. The temperature is no aid. I think that most stress should be laid upon the rash. If obviously scarlatiniform, then I should decide for scarlet fever. It must, however, be kept very clearly in mind that the epiblastic and hypoblastic epithelia are in strong sympathy, of which there are some well-known examples, such as syphilis, scarlet fever, burns, urticaria, variola, pemphigus, not to mention the number of aberrant and fugitive rashes the significance of which is at present unknown. Similar conditions were noted in the last two reports. Compare also the succeeding case.

Ulcer of duodenum.—292. Ulcer on upper surface, close to pylorus; perforation; peritonitis; small yellow infarct in left kidney. Was admitted for general pains and some indefinite swelling of joints. After a few days became covered with raised purpuric rash; urine contained albumen; then diarrhœa, and finally peritonitis.

Abscess of liver. — 305. Chronic peritonitis, chiefly about liver; liver, practically all right lobe, the left being an isolated mass about one inch square. The bile ducts in the upper part are much dilated, and communicate with an abscess cavity about the size of a small egg. This cavity is probably the remains

of the much altered left bile duct. There is also a large abscess between liver and diaphragm, and a diffuse suppuration of the subcutaneous tissue situate over the epigastric region. Clinically the case practically amounted to this, a painful and tender swelling on left part of epigastric region, jaundice, slight pyrexia. The patient, a young woman, had been under my care as out-patient for jaundice in the previous year.

Cancer of stomach.—168. Scirrho-encephaloid; pyloric end; no secondary deposits, outgrowths only. 174. Colloid; pyloric end; secondary deposits in peritoneum. 176. Scirrho-encephaloid; cardiac orifice; secondary deposits in liver, peritoneum, and sigmoid flexure. The sigmoid flexure is thickened and its lumen greatly stenosed by cancerous growth. Microscopical examination shows that the growth is histologically an epithelioma. It is highly probable that the sigmoid flexure was the starting point of the cancer. The clinical aspects of the case were emaciation, persistent diarrhœa, and vomiting. The historical data were dysentery in India, and about eighteen months before admission difficulty of swallowing solid food.

Cancer of intestine.—148. Stricture of descending colon in male, aged 26; peritonitis; ulcerative colitis and enteritis; Meckel's diverticulum. Microscopical examination of the neoplasm shows it to be a columnar epithelioma.

Cancer of bile duct.—132. Large white hard mass springing from gall duct. Its cavity, which is the dilated duct, is choked with gall stones; about 280 were counted in the principal cavity and in its branches in the liver. Numerous masses of softer secondary deposits in liver, which weighed 117 ozs. Microscopical examination showed an ordinary carcinoma. The case during life gave rise to numerous speculations—viz., cystic kidney, hydronephrosis, cancer of kidney, cancer of liver, dilated gall bladder, cyst of peritoneum.

Peritonitis.—179. Peritonitis well marked; pia mater injected and arachnoid surface sticky; convolutions flattened. No obvious cause. I thought at the post-mortem that the condition of brain showed early stage of meningo-cerebritis. The clinical phenomena were delirium, dyspnœa, vomiting, and pyrexia. 290. Peritonitis after premature labour; hydrarthrosis of right knee joint; fibroid quiescent tubercles in lungs.

Lymphatic System.

Chronic tuberculosis of mediastinal glands.—146. Mediastinal glands very large, fibrosed, and caseating; secondary deposits in lungs with excavation; large white mottled kidneys. This case is fully reported and discussed in Westminster Hospital Reports, vol. v. p. 185.

Cancer.—108. Epithelioma of cervical glands. 120. Epithelioma of cervical glands. 273. Epithelioma of glands on right side of neck; considerable ulceration, and also pressure-dislocation of larynx; tracheotomy; pneumonia. The history of the case renders it probable that the disease began in the right tonsil.

Urinary System.

Chronic renal.—128. Weight 5½ and 3½ ozs., granular; hypertrophy of prostate; arteries atheromatous. 138. Female, aged 9. Right kidney 1¾ oz., left ¼ oz.; left in condition of simple general atrophy; right is of a pale pink colour mottled with yellow, and consists of two pyramids with large interpyramidal cortex, the subcapsular cortex being thin. The surface is uneven, but not finely granular. Two ulcers, about the size of a shilling, on lesser curvature of stomach. The upper half of the small intestine is seat of numerous ulcers. In many of these ulcers the floor is formed by peritoneum only. In this case patient was in usual health till six months before admission. Had had "consumptive bowels" at 1½, diphtheria at 2, scarlet fever at 2½, and measles at 5 years of age. 142. Granular; weights, 6¾ and 6 ozs.; hypertrophy of left ventricle. 170. Granular; weights 3¾ and 5¼ ozs.; cortex much diminished; consistence flabby; left ventricle not hypertrophied; chronic pelvic peritonitis. 203. Granular, red, firm; cortex somewhat diminished; weights 2¾ and 3 ozs.; left ventricle greatly hypertrophied; chronic endarteritis. 237. Weights 3½ and 4¼ ozs.; granular; capsules adherent; softish; colour red, mottled with yellow. 130. Weights 7 and 8 ozs.; surface smooth, capsules free; consistence normal; cortex

normal in size; colour dark red, mottled with yellow spots; left ventricle hypertrophied. This patient, female, was under me as out-patient for a long time previous to admission. From the history of the case I connected her disease causally with pregnancy. Was in hospital about five months. The emaciation was extreme. Microscopical examination of kidneys showed that they would be classified with the large white variety; but considering the wasting and other phenomena, the changes in the kidney are astonishingly slight. 166. Weights 8 ozs. each; surface smooth; cortex large, of a yellowish white colour; pyramids purple; heart 5¾ ozs. (patient aged 30); chronic pelvic peritonitis; well-marked hour-glass constriction of stomach, probably congenital. 233. Weights 4½ and 5 ozs.; right kidney reduced to a thin-walled sac containing a white pultaceous mass; left is a large white, contracting; left ventricle hypertrophied.

Generative System.

Pelvic cellulitis.—254. Old and recent pelvic cellulitis; suppurative peritonitis; two fistulous apertures in uterus communicate with abscess cavities in the pelvis. Age of patient 23, and her condition the result of parturition.

Cancer of breast.—289. Scirrhus of left mamma; secondary deposits in many bones, in liver, lymphatic glands, and in right tonsil; no operation. 314. Right mamma absent; secondary deposits in lungs, lymphatic glands, liver, bones, and muscles; uterine myomata and polypi; chronic pelvic peritonitis; left ovary large and cystic.

Organs of Locomotion.

Morbus coxæ.—202. Right hip joint disorganised with numerous sinuses in vicinity; acute pleuritis of both sides; lungs almost solid throughout; several pyæmic abscesses. 218. Left leg wasted and shorter than right; no pus or excess of fluid in or about the left hip joint. On making vertical incision through joint it was found that the ligamentum teres had ulcerated away, that the cartilages of the joint had almost

entirely disappeared, and that the osseous tissue of the femoral head and of the acetabular cavity was red, soft, and crumbly. Large sacral bed sores, with necrosis of coccyx and sacrum, the spinal canal being exposed, and the dura of cord also opened; pus within intermeningeal space, and also diffuse suppuration in front of sacrum. 285. Right leg 3 inches shorter than left. Age of patient 3 years. Fibrous union after excision of hip; caries of ninth and tenth dorsal vertebræ; peritonitis; tubercular pneumonia, and excavation of lung; viscera amyloid.

Injuries and Surgical Operations.

Revolver wound of head.—100. Aperture in skin triangular; in bone circular (external diameter ½ inch, internal ¾ inch). Wound just 1 inch straight above tip of right ear, whence the bullet track passes straight across brain as far as grey matter of left island of Reil, wherein the bullet was found.

Burn.—229, 230, 288. Intestinal mucosa quite normal.

Crushed leg.—144. Left leg swollen and discoloured from extravasation into superficial and deep tissues. Ankle joint contains several drachms of blood-stained fluid. Slight erosion of cartilage on superior surface of astragalus. Several other bruises on body. Patient died with delirium tremens.

Fracture of thigh.—171. Right thigh much swollen and discoloured, and shortened 2 inches. Comminuted fracture of lower end of femur; ends of bone bare, the medulla projecting from both ends as a firm fungating mass. Ends of the bone lie in a cavity filled with bloody fluid. The wall of this sac, which holds about half a pint, is well defined. No attempt at repair. In hospital fifteen days. Liver a well-marked example of cirrhosis; kidneys 12¾ and 10¾ ozs., of a dark purple colour. The size is due to hypertrophy, there being no evidence of structural change. Pia mater thick and opaque, and over anterior half of brain is adherent to cortex.

Excision of jaw.—152. Right inferior maxilla recently removed by surgical operation. Small hole in external carotid just above departure of lingual artery. Suppurative bronchitis.

Gastrostomy.—131. Stomach opened by surgical operation; recent adhesion of anterior surface to abdominal wall. Ulcer-

stricture of œsophagus at level of aortic arch, and just above the level of the tracheal bifurcation. The ulcer is ovalish, about 1 inch in length, with hard shelving edges. At the base of the ulcer are seen several necrosed rings of trachea; but the tracheal mucosa is intact.

Hernia.—123. Left femoral; peritonitis; fibroid tubercles and hæmorrhage into lungs; liver fatty. 133. Umbilical; peritonitis; hydrocele of left tunica vaginalis, and atrophy with fibrosis of left testicle. 200. Left inguinal; peritonitis; perforation of intestine; right inguinal canal patulous. 240. Left inguinal. 263. Left inguinal; peritonitis.

Stricture of urethra.—161. Fistulous openings from urethra into perineum and penis; kidneys "surgical"; calculi in bladder; suppuration of pelvic connective tissue.

Extravasation of urine.—281. Stricture of urethra 3½ inches from meatus; ulcerated hole in urethra just in front of triangular ligament; right kidney is atrophied and cystic, weighs 1¼ oz.; left kidney 6 ozs.

Amputation of penis.—312. Anterior two inches of penis absent; stump quite healed; chronic cystitis; kidneys granular and cystic. Microscopical examination of growth removed at operation shows that it is a papilloma; it consists of finger-like processes of cutis covered with epithelium; within the processes are fibrous tissue and blood-vessels. There is no down growth of epithelium.

Ulcer of leg.—231. Ulcer 10 by 3 inches in front of left leg; subcutaneous tissue in condition of diffuse suppuration; transverse ununited fracture of left upper arm; mesenteric hernia into left scrotum. The hernia comes from the neighbourhood of left kidney, and has no connection with the omentum.

Necrosis of tibia.—216. Necrosis of epiphysis of right tibia, and suppurative arthritis of right knee joint; necrosis of shaft of left tibia.

Ablation of fundus uteri.—121. Absence of left ovary and of uterus above cervix, both of these organs having been removed by surgical operation; right ovary, about size of a hen's egg, is reduced to a thin fibrous sac containing a drab-coloured, pultaceous mass; rupture of bladder; peritonitis; cancer of stomach. 195. Both ovaries and uterus above cervix absent,

having been removed by surgical operation; emphysema; fatty heart.

Ablation of uterine myoma.—109. Stump situated at top of fundus uteri; much blood and clot in peritoneal sac; peritonitis; ovaries large, hard, and cystic.

Ovariotomy.—296. Surgical wound communicates directly with large cyst of right ovary, which is inseparably adherent to wall of abdomen in front and flanks; peritonitis.

Unclassified.—198. This case was fully reported in Westminster Hospital Reports, vol. v. p. 195. The post-mortem appearances were practically negative. In the remarks on this case it is suggested that the patient died from shock after the administration of an enema. 239. This patient was a child, aged 6, who died in a few hours after admission. The post-mortem appearances were for the most part negative. The only positive facts of importance were that the convolutions of the brain, which weighed 50½ ozs., were flattened, that the pia mater was more or less adherent everywhere, and that the cerebral substance was less consistent than usual. There was, however, no hyperæmia either of the nervous matter or of the membranes. There was no excess of fluid either in brain or spinal cord. If I remember correctly, the principal symptoms were pyrexia and delirium. The child's father was in the hospital at the time for lead palsy, and there was an ill-verified statement that the child had eaten some whitelead a few days before admission. Upon this a suggestion was made that the case might be acute plumbism with cerebral phenomena. No lead was found in the viscera on chemical examination.

MEDICAL REGISTRAR'S REPORT FOR THE YEAR 1889.

By Dr. W. A. WILLS.

Number of patients in Hospital, Jan. 1st, 1889		77
" " " Dec. 31st, 1889		80
Admitted to Medical Wards during the year		1146
Of these—Males	628	
Females	518	
	—	1146
Average number of patients in Hospital		70·23
Average stay in Hospital of each patient		22·37 days
Recovered or relieved:		
Males	508	
Females	428	
	—	936
Unrelieved, including those sent to Surgical or Obstetrical Wards:		
Males	28	
Females	28	
	—	56
Died:		
Males	92	
Females	62	
	—	154

Table A.

Disease.	Jan. to March.	April to June.	July to Sept.	Oct to Dec.	Sex. Males.	Sex. Females.	Sex. Total.	Age. Under 5 yrs.	Age. 5—10.	Age. 10—20.	Age. 20—30.	Age. 30—40.	Age. 40—50.	Age. 50—60.	Age. Over 60.	Result. Death.	Result. Unrelieved.	Result. Relieved.	Remarks.
General Diseases:																			
Group A:																			
Scarlatina	...	1	2	...	2	1	3	1	...	2	...	...	...	...	...	...	2	1	
Morbilli	7	6	1	...	9	5	14	10	1	1	2	...	...	...	...	2	1	11	
Rötheln	1	...	1	...	1	2	3	1	1	1	...	...	...	...	...	...	...	3	
Enteric fever	...	3	6	9	9	9	18	...	4	6	7	...	...	...	...	2	...	16	*vide* Table B
Diphtheria	12	1	4	4	12	9	21	6	12	3	...	...	...	...	...	8	...	13	
Febricula	4	3	2	5	6	8	14	11	...	2	1	...	...	...	...	...	...	14	
Varicella	1	...	...	...	1	...	1	1	...	...	...	...	...	...	...	1	...	...	
Erysipelas	5	2	...	1	3	5	8	2	...	1	1	3	1	...	...	...	...	8	
Pertussis	4	1	...	4	4	5	9	9	...	...	...	...	...	...	...	...	2	7	
Syphilis	...	3	...	...	1	2	3	...	...	...	1	1	...	...	1	...	2	3	
Intermittent fever	1	...	...	1	2	...	2	...	...	...	..	2	...	...	...	...	...	2	
Tetanus	...	...	1	...	1	...	1	...	1	...	...	...	...	...	...	...	...	1	
Influenza	...	...	...	3	3	...	3	...	...	...	2	1	...	...	...	...	...	3	
Group B:																			
Poisons:																			
Alcohol	...	1	...	1	1	1	2	...	...	...	...	2	...	...	...	...	...	2	
Belladonna	...	...	...	1	1	...	1	1	...	...	...	...	...	...	...	...	...	1	
Strychnine	...	1	...	...	...	1	1	...	...	...	...	1	...	...	...	...	...	1	
Lead	4	3	2	4	13	...	13	...	...	...	3	4	6	...	...	...	...	13	
Arsenic	...	...	...	1	1	...	1	...	...	...	...	...	1	...	...	...	...	1	
Paraffin	...	1	...	...	1	...	1	...	1	...	...	...	...	...	...	...	...	1	
Group C:																			
Debility	...	...	...	2	2	...	2	...	...	1	...	...	...	...	1	...	...	2	
Group D:																			
Not classified:																			
Acute Rheumatism	15	12	15	11	29	24	53	...	1	15	25	7	5	...	...	5	...	48	*vide* Table C
Subacute ditto	...	...	7	4	7	4	11	...	...	2	4	1	2	2	...	...	...	11	
Chronic ditto	4	9	3	1	6	11	17	...	...	3	6	5	2	1	...	...	...	17	
Gout	...	...	1	1	1	1	2	...	...	...	...	1	...	1	...	...	...	2	
Tuberculosis	4	5	2	3	3	11	14	9	2	1	2	...	...	...	...	9	...	5	
Rickets	1	3	1	...	3	2	5	4	...	1	...	...	...	...	...	...	1	4	
Anæmia	17	2	5	8	...	32	32	...	...	15	13	3	1	...	...	...	...	32	
Diabetes	2	...	...	1	2	1	3	...	...	...	1	...	...	2	...	...	...	3	
Myxœdema	1	1	...	1	...	3	3	...	...	...	...	2	...	1	...	...	...	3	
Purpura	1	1	...	...	...	2	2	...	...	...	...	1	...	1	...	...	...	2	
Hæmophilia	...	...	...	1	1	...	1	...	...	...	1	...	...	...	...	...	...	1	
Diseases of the Nervous System:																			
Brain:																			
Hæmorrhage	...	...	...	1	...	1	1	...	...	1	...	...	...	...	...	1	...	...	
Hemiplegia	6	1	3	2	5	7	12	...	...	...	2	1	3	5	1	...	1	11	
Tumour	...	2	...	1	2	1	3	...	1	1	...	...	1	..	...	2	...	1	

TABLE A—*continued.*

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex. Males.	Sex. Females.	Sex. Total.	Age. Under 5 yrs	Age. 5—10.	Age. 10—20.	Age. 20—30.	Age. 30—40.	Age. 40—50.	Age. 50—60.	Age. Over 60.	Result. Death.	Result. Unrelieved.	Result. Relieved.	Remarks.
DISEASES OF THE NERVOUS SYSTEM—*continued*—																			
Hydrocephalus	1	...	...	2	3	...	3	3	...	...	...	...	...	...	...	2	...	1	
Abscess	...	1	...	...	...	1	1	...	...	...	...	1	...	...	...	1	...	...	
Meningitis	1	...	2	1	4	...	4	2	1	...	...	1	...	...	...	4	...	...	
Cerebro-spinal sclerosis	1	...	1	...	1	1	2	...	...	...	1	1	...	...	...	...	1	1	
Concussion	1	...	...	2	3	...	3	1	...	...	1	1	...	...	...	...	...	3	
Cerebritis	...	...	1	...	1	...	1	...	1	...	...	...	...	...	...	1	...	...	
Cord:																			
Acute myelitis	...	1	...	...	1	...	1	1	...	...	...	...	...	...	...	...	...	1	
Anterior polio-myelitis	4	...	1	...	2	3	5	3	1	1	...	...	...	...	...	...	2	3	
Spastic paralysis	...	...	...	2	2	...	2	...	...	1	...	...	...	1	...	...	...	2	
Tabes dorsalis	3	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
Progressive muscular atrophy	1	...	...	1	2	...	2	...	...	...	...	2	...	1	...	...	1	1	
Meningitis	...	...	1	...	1	...	1	1	...	...	...	...	...	...	...	1	...	...	
Sciatica	5	...	2	...	7	...	7	...	...	1	...	1	1	3	1	...	1	6	
Traumatic neuritis	1	1	1	...	3	...	3	...	...	1	2	...	...	...	...	...	...	3	
Diphtheritic paralysis	1	1	...	...	1	1	2	1	1	...	...	...	...	...	...	...	...	2	
Alcoholic paralysis	...	...	...	1	...	1	1	...	...	...	...	...	1	...	...	...	...	1	
Migraine	1	...	1	...	1	1	2	...	...	...	...	1	...	1	...	...	...	2	
Epilepsy	3	1	1	...	3	2	5	...	..	3	1	...	...	...	1	...	...	5	
Infantile convulsions	1	3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
Chorea	1	2	5	5	3	10	13	...	2	10	1	...	...	...	...	...	...	13	*vide* Table D
Hysteria	10	3	7	8	6	22	28	...	...	8	13	6	..	1	...	...	3	25	
Hystero-epilepsy	4	1	...	...	2	3	5	...	...	...	4	...	1	...	...	...	1	4	
Mental:																			
Mania	...	1	...	...	...	1	1	...	...	...	...	1	...	...	...	...	1	...	
General paralysis	2	3	...	...	2	3	5	...	...	...	...	2	2	1	...	...	4	1	
Delirium tremens	1	...	1	1	3	...	3	...	...	1	2	...	...	...	...	2	...	1	
Idiocy	...	...	...	1	...	1	1	...	...	1	...	...	...	...	...	...	1	...	
DISEASES OF THE CIRCULATORY SYSTEM:																			
Dilatation of heart	1	2	2	1	3	3	6	...	...	...	1	...	2	...	3	3	...	3	
Endocarditis (not arising in hospital):																			
Mitral:																			
1. With presystolic murmur	...	1	...	1	...	2	2	...	...	...	...	...	2	...	...	...	...	2	
2. With systolic murmur	4	5	5	6	9	11	20	...	2	9	3	1	2	2	1	...	1	19	
3. With double murmur	10	7	8	8	16	17	33	1	2	10	11	7	1	1	...	13	...	20	

TABLE A—*continued.*

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex. Males.	Sex. Females.	Sex. Total.	Age. Under 5 yrs.	Age. 5—10.	Age. 10—20.	Age. 20—30.	Age. 30—40.	Age. 40—50.	Age. 50—60.	Age. Over 60.	Result. Death.	Result. Unrelieved.	Result. Relieved	Remarks.
DISEASES OF THE CIRCULATORY SYSTEM—*continued*—																			
Aortic:																			
1. With systolic murmur	...	3	...	...	3	...	3	...	...	...	...	1	2	...	...	...	...	3	
2. With diastolic murmur	...	1	...	...	1	...	1	...	...	...	...	..	...	1	...	...	1	...	
3. With double murmur	5	4	5	5	17	2	19	...	...	...	7	6	1	3	2	3	...	14	
Pulmonary:																			
With double murmur	2	1	...	1	1	3	4	...	1	1	1	...	1	...	...	1	...	3	
Pericarditis	3	1	1	...	4	1	5	1	1	...	2	1	...	...	...	1	...	4	
Cyanosis	...	1	1	...	2	...	2	1	...	...	1	...	...	...	...	...	...	2	
Aneurism:																			
1. Thoracic	2	1	1	1	5	...	5	1	1	...	2	1	...	...	...	1	...	4	
2. Abdominal	1	1	...	...	1	1	2	...	...	...	...	...	...	1	1	...	...	2	
Angina pectoris	2	...	1	...	3	...	3	...	...	...	1	...	...	...	2	...	...	3	
Phlegmasia dolens	1	2	1	1	...	5	5	...	...	...	2	2	1	...	...	...	...	5	
Diseases of the thyroid gland:																			
Malignant disease	...	...	...	1	...	1	1	...	...	...	...	...	...	1	1	..	...	...	
Exophthalmic goitre	...	...	...	1	...	1	1	...	...	1	...	...	...	...	...	...	...	1	
DISEASES OF THE RESPIRATORY SYSTEM:																			
Laryngitis	5	1	...	3	5	4	9	5	2	1	1	...	...	...	...	...	...	9	
Tubercular disease of larynx	1	...	...	...	1	...	1	...	...	...	...	1	...	...	...	...	...	1	
Malignant disease of larynx	...	1	...	...	1	...	1	...	...	...	...	...	...	1	...	...	1	...	
Bronchitis:																			
1. Acute	13	14	9	12	24	24	48	34	8	2	...	2	...	1	1	1	...	47	
2. Chronic	18	5	5	12	23	17	40	1	1	3	2	5	10	8	10	9	...	31	
Pneumonia:																			
1. Lobar	18	10	8	15	33	18	51	8	4	10	9	8	5	6	1	16	...	35	
2. Lobular	11	11	12	9	25	18	43	36	7	...	...	...	...	...	...	12	1	30	
Pleurisy:																			
1. Without effusion	2	3	2	4	6	5	11	...	...	2	4	3	2	...	...	...	1	10	
2. With effusion	5	3	2	2	11	1	12	2	...	3	2	...	2	2	...	1	...	11	
3. Empyema	2	...	2	2	2	4	6	3	...	1	1	...	1	...	...	1	...	5	
Phthisis	30	12	24	26	61	31	92	...	5	13	24	25	19	4	2	11	7	74	
Emphysema	1	2	1	...	2	2	4	...	...	...	...	1	1	1	1	1	...	3	
Mediastinal tumour	3	...	...	2	5	...	5	...	...	1	1	1	1	...	1	...	1	4	
Asthma	1	1	1	...	2	1	3	...	...	1	2	...	...	...	...	...	...	3	

TABLE A—*continued.*

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex.			Age.								Result.			Remarks.
					Males.	Females.	Total.	Under 5 yrs.	5—10.	10—20.	20—30.	30—40.	40—50.	50—60.	Over 60.	Death.	Unrelieved.	Relieved.	
DISEASES OF THE DIGESTIVE SYSTEM:																			
Mouth:																			
Stomatitis	1	...	...	2	2	1	3	3	...	...	...	...	...	...	...	...	...	3	
Epithelioma of fauces	1	...	...	1	2	...	2	...	...	...	...	...	...	2	...	1	1	...	
Pharynx and Œsophagus:																			
Tonsillitis:																			
1. Acute	6	14	16	13	29	20	49	9	4	26	26	4	...	...	...	...	...	49	
2. Chronic	...	...	1	...	1	...	1	...	...	...	1	...	...	...	...	...	...	1	
Œsophagal stricture:																			
1. Functional	...	...	...	1	1	...	1	...	...	...	...	...	...	1	...	...	...	1	
2. Malignant	...	1	...	...	...	1	1	...	...	...	...	...	...	1	...	...	...	1	
Stomach:																			
Dyspepsia	1	4	1	...	...	6	6	...	...	3	1	...	1	1	...	...	1	5	
Acute gastritis	2	...	...	...	2	...	2	...	...	...	...	1	...	1	...	...	...	2	
Ulcer	7	2	5	6	3	17	20	...	...	2	10	7	1	...	...	...	...	20	
Carcinoma	1	2	1	...	3	1	4	...	...	...	...	...	1	1	2	3	...	1	
Intestines:																			
Diarrhœa	4	12	8	8	19	13	32	24	2	3	...	1	...	1	1	9	3	20	
Constipation	4	1	1	3	3	6	9	...	1	3	...	2	1	1	1	1	...	8	
Colic	1	3	2	1	5	2	7	1	...	1	...	2	1	2	...	...	...	7	
Typhlitis	...	3	1	4	5	3	8	...	1	4	2	1	...	...	...	1	...	7	
Enteritis	1	...	4	...	2	3	5	1	...	1	1	...	1	...	1	1	...	4	
Perforation of Vermiform Appendix	...	...	...	1	1	...	1	...	1	...	...	...	...	...	...	1	...	...	
Malignant disease	2	...	...	...	1	1	2	...	...	...	1	...	...	...	1	1	...	1	
Dysentery	...	...	1	...	1	...	1	...	...	...	1	...	...	...	...	1	...	...	
Duodenal ulcer	...	...	...	1	1	...	1	...	...	1	...	...	...	...	...	1	...	...	
Liver:																			
Jaundice	2	3	2	...	3	4	7	...	...	2	...	1	2	...	2	...	...	7	
Gall stone colic	3	...	1	...	2	2	4	...	...	...	1	1	...	1	1	...	...	4	
Cirrhosis	3	6	2	1	8	4	12	...	...	...	4	4	3	1	...	...	...	12	
Carcinoma	2	...	1	...	1	2	3	...	...	...	...	...	1	...	2	2	...	1	
Hydatid tumour	...	1	...	...	...	1	1	...	...	...	1	...	...	...	...	...	...	1	
Abscess	...	...	...	1	...	1	1	...	...	1	...	...	...	...	...	1	...	...	
Pancreas:																			
Malignant disease	...	...	1	...	1	...	1	...	...	...	...	...	...	...	1	1	...	...	
Peritoneum:																			
Peritonitis:																			
1. Acute	1	2	2	...	3	2	5	...	1	2	1	1	...	...	...	1	...	4	
2. Chronic	2	...	...	...	2	2	1	...	1	...	...	...	...	...	...	...	...	2	
Ascites	2	1	1	1	2	3	5	...	...	...	...	1	1	...	3	...	1	4	

TABLE A—*continued.*

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex. Males.	Sex. Females.	Sex. Total.	Age. Under 5 yrs.	Age. 5—10.	Age. 10—20	Age. 20—30.	Age. 30—40.	Age. 40—50.	Age. 50—60.	Age. Over 60.	Result. Death.	Result. Unrelieved.	Result. Relieved.	Remarks.
DISEASES OF THE LYMPHATIC SYSTEM:																			
Splenic leucocythæmia	...	1	1	1	3	...	3	...	...	...	...	1	...	...	2	1	...	2	
Addison's disease	...	2	...	1	3	...	3	...	...	1	...	...	2	...	...	...	...	3	
DISEASES OF THE URINARY SYSTEM:																			
Acute nephritis	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
Chronic nephritis:																			
1. Large white	...	1	3	3	4	3	7	1	1	...	1	...	3	...	1	...	...	7	
2. Contracted	5	3	6	3	11	6	17	1	1	2	5	2	4	2	...	1	...	16	
3. Suppurative	1	...	...	...	...	1	1	...	...	...	...	1	...	...	...	1	...	...	
Tubercular disease	...	1	...	...	1	...	1	...	...	...	...	1	...	...	...	...	1	..	
Renal calculus	1	1	1	2	4	1	5	...	...	...	...	2	2	1	...	...	1	4	
Paroxysmal hæmoglobinuria	...	2	...	...	2	...	2	...	...	...	...	...	2	...	...	...	...	2	
Malignant disease of bladder	1	...	...	...	1	...	1	...	...	...	...	...	...	...	1	...	1	...	
Cystitis	...	...	2	...	...	2	2	...	...	...	...	1	...	...	1	...	1	1	
Floating kidney	...	...	...	1	...	1	1	...	...	...	...	...	...	1	...	...	...	1	
DISEASES OF THE SKIN:																			
Herpes labialis	...	1	...	1	1	1	2	...	...	2	...	...	...	...	...	...	...	2	
Herpes zoster	2	...	...	...	1	1	2	...	1	1	...	...	...	...	...	...	...	2	
Eczema	...	...	1	...	...	1	1	...	...	...	...	...	...	...	1	...	...	1	
Pemphigus	1	1	1	1	...	4	4	2	...	...	1	...	1	...	...	...	...	4	
Urticaria	1	...	...	...	1	...	1	...	...	...	...	1	...	...	...	...	...	1	
Erythema nodsum	...	1	...	...	...	1	...	1	...	...	1	...	...	...	...	...	...	1	
DISEASES OF THE GENERATIVE SYSTEM:																			
Gonorrhœa	1	...	...	...	1	...	1	...	...	1	...	...	...	...	...	...	...	1	
Leucorrhœa	1	...	...	1	...	2	2	...	...	1	...	1	...	...	...	...	...	2	
DISEASES OF WOMEN:																			
Fibroid of uterus	2	...	...	...	...	2	2	...	...	...	...	...	1	...	1	...	1	1	
Subinvolution	...	1	...	...	...	1	1	...	...	...	...	1	...	...	...	...	1	...	
Pelvic cellulitis	2	...	...	...	...	2	2	...	...	...	...	...	2	...	...	...	1	1	
Uterine complaints (transferred to Arden)	...	...	...	1	...	1	1	...	...	...	1	...	...	...	...	...	1	...	
VARIOUS:																			
Pregnancy	2	...	...	...	...	2	2	...	...	...	1	...	1	...	...	...	2	...	
Marasmus	3	2	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
Hæmorrhage from the bowel	1	...	...	...	1	...	1	1	...	...	...	...	...	...	...	...	...	1	
Conjunctivitis	1	...	...	...	...	1	1	1	...	...	...	...	...	...	...	...	...	1	
Abscesses (various)	1	2	1	...	1	3	4	1	...	...	2	...	1	...	...	...	1	3	
Improper feeding	...	3	...	...	3	...	3	3	...	...	...	...	...	...	...	...	...	3	

TABLE A—*continued.*

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex.			Age.								Result.			Remarks.
					Males.	Females.	Total.	Under 5 yrs.	5—10.	10—20.	20—30.	30—40.	40—50.	50—60.	Over 60.	Death.	Unrelieved.	Relieved.	
VARIOUS—*continued*—																			
Wry neck	...	1	...	1	1	1	2	...	1	...	...	...	...	1	...	...	...	2	
Lumbago	...	1	...	...	1	...	1	...	...	...	...	...	1	...	...	...	...	1	
Diabetes insipidus	...	1	...	1	1	1	2	...	...	...	...	1	...	...	1	...	1	1	
Heat stroke	...	...	1	...	1	...	1	...	...	...	...	1	...	...	...	...	...	1	
Giddiness	...	1	...	...	1	1	1	...	...	...	...	...	...	...	1	...	...	1	
Deafness	...	...	1	...	1	...	1	...	...	...	1	...	...	...	...	...	1	...	
Incontinence of Urine	...	...	...	1	1	...	1	1	...	...	...	...	...	...	...	...	...	1	
Injury to rib	...	...	...	1	1	...	1	...	...	...	...	...	...	...	1	...	...	1	
Diffuse lipomata	...	...	...	1	...	1	1	...	...	...	...	...	...	1	...	...	...	1	
Malingering	1	...	...	1	1	1	2	...	...	...	...	1	1	...	...	...	...	2	
Nil	2	4	1	1	4	4	8	1	3	3	1	...	...	...	...	...	...	8	

TABLE B.—*Enteric Fever.*

Eighteen cases of Enteric Fever were treated in the hospital during the year 1889.

The average stay in the hospital of each patient was 32·94 days.

There were two deaths, both female patients. Both deaths occurred soon after profuse hæmorrhage from the bowel, although in one case the hæmorrhage appeared to be largely due to blood coming from the nose which had been swallowed. Both cases were admitted in the second week, and had been walking about until admission. Only four cases this year suffered from constipation throughout the illness. Two cases suffered relapse.

In two instances two or more persons from the same household were admitted with enteric fever at a short interval of time. In one of these instances four persons (including one case in 1890) were attacked.

In eight cases the drains of the houses inhabited by the patients were stated to be out of order.

TABLE B—*continued.*

No. and Name.	Month admitted.	Sex.	Age.	Onset.	Character.	Fever.	Days in hosp.	Result.	Treatment.	Remarks.
1 T. R.	April	M.	6	Headache, sickness, diarrhœa, restlessness at night	Admitted on 12th day; abdomen slightly distended; a few spots; spleen enlarged; some bronchitis; diarrhœa about 6 times daily, gradually getting less	11 days' fever; highest temp. 104·4°	43	Recov.	—	Very slow convalescence.
2 T. B.	May	M.	12	Sudden vomiting diarrhœa, and pain in left iliac region	Admitted on 3rd day; slight case; no distension of abdomen; a few doubtful spots; bronchitis; diarrhœa; motions light	4 days' fever; highest temp. 101·4°	—	"		
3 S. S.	"	F.	18	Gradual sickness, pains in limbs, epistaxis	Admitted on 12th day; abdomen distended; tender; spots; spleen enlarged; bronchitis severe; soft apex systolic murmur; diarrhœa at first, then motions formed; tremulous. On 23rd day profuse epistaxis, and a few hours later melæna: recurred frequently during the next 2 days; abdomen became much distended, and she gradually became collapsed and died	14 days' fever; highest temp. 106·2°	14	Death	Spongeing at 103·0°, 105·2°, 105·6°, 105·8°. Wet pack at 106°	Walking about until admission. A lady in the house where she was servant was ill with enteric fever. P.M.—A few deep ulcers in cæcum; retroperitoneal glands much enlarged: much bronchitis; heart and kidneys pallid. The bowel hæmorrhage apparently did not come from the intestinal ulceration.

4 B. Y.	July	F.	24	Headache, anorexia, diarrhœa, epistaxis	Admitted on 9th day; abdomen distended; spots; delirious; much diarrhœa. Profuse hæmorrhage from bowel on 15th day	11 days' fever; highest temp. 105·3°	11	"	Spongeing, brandy, opium, hazeline, ergotine	Walking about until admission. P.M.—42 inches of the ileum showed typhoid ulceration and swelling of the glands, the latter being more marked.
5 E. W.	"	F.	22	Ailing for 4 or 5 weeks. Weakness, anorexia	Admitted on 10th day; severe attack; delirious at nights; abdomen distended; spots; diarrhœa; motions dark	10 days' fever; highest temp. 104·4°	26	Recov.	—	In the rectum, 4 inches from the anus, were 4 deep punched out ulcers.
6 F. P.	Aug.	F.	22	Sudden headache, vomiting, shivering, anorexia	Admitted on 10th day; very slight case	6 days' fever; highest temp. 103°	22	"		
7 B. N.	"	F.	26	Headache, pain in abdomen, diarrhœa	Admitted on 8th day; slight abdominal distension; spots; tongue furred and tremulous; delirious, afterwards becoming actively maniacal. Good convalescence	9 days' fever; highest temp. 104°	29	"	Spongeing, pot. bromid., chloral hyd., hyoscine	
8 F. G.	"	F.	25	Weakness, pains in limbs	Admitted on 8th day; spots; constipation. Slight case	10 days' fever; highest temp. 103·4°	27	"	Acid hydrochl. dil., enema, glycerine	Working up to day of admission. Bad drains in house
9 W. C.	Sept.	M.	34	Headache, sickness, anorexia	Admitted on 7th day; abdomen distended; no spots; tongue furred and dry; considerable prostration and slow convalescence	4 days' fever; highest temp. 100°	34	"		
10 A. J.	Oct.	M.	16	Gradual, anorexia, cough, weakness, diarrhœa. Motions and urine passed in bed	Admitted on 14th day; very prostrate; slight diarrhœa; spots; bronchitis. Otorrhœa during convalescence, which was slow but good	24 days' fever; highest temp. 105·4°	65	"		

TABLE B—*continued.*

No. and Name.	Month admitted.	Sex.	Age.	Onset.	Character.	Fever.	Days in hosp.	Result.	Treatment.	Remarks.
11 A. A.	Oct.	F.	15	Drowsiness, headache	Admitted on 5th day; abdomen distended; spots; constipation. Good convalescence	14 days' fever; highest temp. 103·8°; rose to 99·6° a week after. Subsidence owing to constipation	37	Recov.	Enemata	From same house as No. 13.
12 W. G.	"	M.	28	Sudden sickness and diarrhœa, headache, weakness	Admitted on 19th day; abdomen distended; spots; diarrhœa; motions light; very prostrate; pulse dicrotic; slight epistaxis. Recrudescence on 14th day; tongue furred, dry; spots; bronchitis; ? collapse of base of left lung. During convalescence pulsation suddenly ceased in left femoral artery for some days, apparently from pressure of enlarged femoral glands. He had 2 rigors. Pulsation gradually returned	40 days' continuous fever; temp. falling almost to normal on the 14th day (highest temp. 104·6°), but at once rising gradually again to 104·4° in 5 days. This second period of fever continued for 16 days, gradually falling to normal; but it again rose to 102°, apparently owing to the leg condition. In the rigors temp. rose to 104° & 104·8°	68	"	Brandy, pulv. ipec. co., ext. opii, quinine	He smelt a very bad smell in Dublin on the 7th October, at a house where a man had just had enteric fever. He was at once sick, and his illness then commenced.

13 M. A.	„	F.	7½	Gradual onset, with general malaise for 3 or 4 weeks before admission	Admitted at end of 2nd week; numerous spots; tongue dry and furred; constipation; slight delirium at night	12 days' fever; highest temp. 103·6°	41	„	—	From same house as No. 11.
14 G. G.	Nov.	M.	27	Weakness, pains in limbs, and headache	Admitted at beginning of 3rd week; abdomen distended; spots; diarrhœa at first, afterwards constipation. Memory much impaired at first. Systolic apex murmur. Relapse on 21st day; diarrhœa; spots; abdomen distended. Good convalescence	13 days' fever; highest temp. 103·4°. In relapse, 13 days' fever; highest temp. 101·4°	36	„	Brandy	
15 T. B.	„	M.	11	Sudden headache, nausea, pain in abdomen	Admitted on 3rd day; abdomen distended, tender; spots numerous; bowels regular; bronchitis; considerable prostration; deafness Slight epistaxis. Mitral regurgitation. Good convalescence	24 days' fever; highest temp. 104·6°	50	„	—	Admitted a month previous with an attack of either typhlitis or enteric fever, but temp. had been normal for 21 days before readmission. Erlich's test of sulphanilic acid in presence of free nitrous acid gives red coloration of urine. Previously in hospital for chorea, when he was noted to have a mitral murmur. Discharged in January, 1890.
16 J. P.	Dec.	M.	10	Ailing for some little time	Admitted on 5th day; abdomen distended; spleen enlarged; spots; bronchitis; bowels regular. Drowsy all through attack	17 days' fever; highest temp. 105°	40	„	Sponged	Came from Hendon, where there is much enteric fever. From same house as Nos. 17 & 18, his brother and sister. Discharged in January, 1890.

TABLE B—*continued.*

No. and Name.	Month admitted.	Sex.	Age.	Onset.	Character.	Fever.	Days in hosp.	Result.	Treatment.	Remarks.
17 W. P.	Dec.	M.	6	Headache, abdominal pain	Admitted on 4th day; abdomen distended and very tender; spleen enlarged; drowsy. Constantly crying with pain in abdomen. Motions formed	13 days' fever; highest temp. 102·8°	28	Recov.	—	From same house as Nos. 16 & 18, where the drain has been completely blocked. A person in a neighbouring house has had enteric fever recently. Erlich's test gives a red coloration. Discharged in January, 1890.
18 E. P.	,,	F.	8	Headache, drowsiness, pain in abdomen, constipation	Admitted on 2nd day; slight case; abdomen slightly distended; tongue furred, with red papillæ; constipation	13 days' fever; highest temp. 103·4°	21	,,	—	From same house as Nos. 16 & 17. Erlich's test gives a red coloration. Discharged in January, 1890.

TABLE B—*continued.*

No. and Name.	Month admitted.	Sex.	Age.	Onset.	Character.	Fever.	Days in hosp.	Result.	Treatment.	Remarks.
17 W. P.	Dec.	M.	6	Headache, abdominal pain	Admitted on 4th day; abdomen distended and very tender; spleen enlarged; drowsy. Constantly crying with pain in abdomen. Motions formed	13 days' fever; highest temp. 102·8°	28	Recov.	—	From same house as Nos. 16 & 18, where the drain has been completely blocked. A person in a neighbouring house has had enteric fever recently. Erlich's test gives a red coloration. Discharged in January, 1890.
18 E. P.	,,	F.	8	Headache, drowsiness, pain in abdomen, constipation	Admitted on 2nd day; slight case; abdomen slightly distended; tongue furred, with red papillæ; constipation	13 days' fever; highest temp. 103·4°	21	,,	—	From same house as Nos. 16 & 17. Erlich's test gives a red coloration. Discharged in January, 1890.

TABLE C.—*Acute Rheumatism.*

In last year's Report a summary was given of all the cases of acute rheumatism treated in the Hospital during the years 1881-2-3-4-5-6-7-8, with especial reference to the following points: age, sex, months, seasons of the year, duration of attack, heart complications, and inheritance.

To this summary are now added the details of the cases treated during the year 1889, the total number of cases being 609.

Ages.

	Under 5.	5—10.	10—20.	20—30.	30—40.	40—50.	50—60.	Over 60.
1881	0	2	31	22	23	5	1	0
1882	1	3	29	16	12	5	1	0
1883	0	1	18	28	14	8	2	1
1884	0	16	33	29	14	5	3	0
1885	0	1	26	21	12	1	1	1
1886	0	0	17	13	4	2	0	0
1887	0	2	29	24	10	3	1	0
1888	1	5	18	29	9	3	0	0
1889	0	1	15	25	7	5	0	0
Total	2	31	216	207	105	37	9	2

Sex.

	Males.	Females.
1881	43	41
1882	25	42
1883	39	33
1884	46	54
1885	31	32
1886	13	23
1887	34	35
1888	38	27
1889	29	24
Total	298	311

Numbers admitted in each Month.

January	62	July	34
February	56	August	47
March	53	September	64
April	45	October	64
May	41	November	42
June	44	December	57

Seasons.

Cases admitted, in each group of three months.

January to March	171	July to September.....	146
April to June	130	October to December...	162

Cases admitted, in two groups: six warmer and six colder months.

April to September ...	269	October to March ...	340

Average stay in Hospital—25·38 days.

Inheritance.

	Rheumatism.	Gout.	Neurosis.	Phthisis.
1881	26	7	3	11
1882	34	2	13	19
1883	32	6	9	9
1884	42	14	22	8
1885	17	7	22	18
1886	11	2	3	5
1887	28	0	8	14
1888	37	3	0	9
1889	18	1	6	19
Total.....	245	42	86	112

Character.

Months.	No. of Cases.	Average stay in days.	No. of Cases with heart disease.	Character of heart disease.		No. of Cases admitted with old heart disease.
				Peri-carditis.	Endo-carditis.	
January	62	30·77	36	11	33	9
February	56	30·38	37	20	26	11
March	53	29·62	28	13	24	6
April	45	34·57	25	8	22	11
May	41	28·64	21	12	18	6
June	44	27·55	24	10	18	8
July.....	34	20·60	20	6	14	10
August	47	20·93	23	10	15	8
September	64	25·90	30	9	21	12
October	64	31·53	37	13	28	12
November	42	25·15	31	12	14	6
December	57	25·09	31	11	20	12

The whole number of cases with heart affection is 343; of these 135 suffered from pericarditis, and 253 from endocarditis. The number includes 111 cases with old heart disease, as shown by cardiac bruits, and 78 who had both pericarditis and endocarditis. The number leaving Hospital with persistent bruits is 261.

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TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart. Affection.	Heart. In which attack developed.	Heart. State on discharge.	No. of days in hospital.	Result.	Treatment	Remarks.
1 C. F.	M.	20	Jan.	6th	3	17	Father suffers from rheumatism, mother has dropsy	4	3	101°	Knees, ankles	Endocarditis	Previous	Double aortic and mitral regurgitant murmurs	16	Recovery	Hst. sod. sal.	In hospital last year with acute rheumatism.
2 S. R.	M.	27	„	7th	0	27	Mother and a sister died of consumption	3	9	100·6°	All large joints	Endocarditis	Present	Systolic at apex	21	„	„	Slight albuminuria.
3 C. C.	F.	17	„	3rd	1	17	1 brother suffers from rheumatism, father has consumption	5	5	100·0°	Left knee, spine	Soft systolic at apex	„	Systolic at apex, apex in normal position	16	„	„	Re-admitted 14 days after discharge in 1888.
4 E. A. A.	F.	33	„	14th	0	—	Father died æt. 30 of consumption, a sister died æt. 15 of hip disease	19	14	103·4°	Both wrists, fingers, left ankle	Pericarditis; soft systolic at apex	„	Occasional systolic at apex, apex in normal position	39	„	Hst. sod. sal., pulv. ipec. co., quinin. sulph.	Rheumatic pains 16 years ago. She was admitted with symptoms of perforation from gastric ulcer; these disappeared after 3 days. Then slight swelling of the joints, and sub-conjunctival hœmorrhages appeared,

																		which were thought to be pyæmic; gradually developed into an undoubted case of acute rheumatism.
5 J. W.	M.	41	„	3rd	4	29	1 brother suffering from paraplegia	12	12	104°	Spine, arms, knees, shoulders, right wrist	Pericarditis, endocarditis	Previous and present	Systolic murmur at apex, apex outside nipple	29	„	Hst. sod. sal.	A carman, often gets wet. Very profuse sweating.
6 T. H.	M.	24	„	3rd	2	17	—	2	4	100·4°	Ankles, knees, hips	Endocarditis	Previous	Mitral regurgitation	19	„	„	Was in hospital in Dec., 1888. Murmur much more evident since then.
7 A. S.	M.	14	Feb.	14th	1	10	—	3	2	100·8°	Shoulders, fingers	Endocarditis	„	Mitral regurgitation, apex outside nipple	19	„	„	—
8 J. L.	M.	22	„	7th	1	15	Father died of consumption, mother has epileptic fits	22	14	100·6°	Ankles, feet	Endocarditis	Previous and present	Mitral regurgitation and double aortic	67	„	„	Admitted with hæmaturia and much albumen, which afterwards cleared up.
9 P. R.	F.	18	„	7th	0	0	—	8	12	104·0°	Ankles, elbows, shoulders	Pericarditis	Present	Occasional systolic murmur at apex	47	„	„	Had suffered from rheumatic pains before.
10 H. D.	M.	19	„	14th	0	0	Mother has epileptic fits	14	10	103·4°	Ankles, spine, left hand	Pericarditis, endocarditis	„	Mitral regurgitation, apex in nipple line	48	„	„ brandy	Severe attack, delirious at nights. Hypostatic congestion of lungs.

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart. Affection.	Heart. In which attack developed.	Heart. State on discharge.	No. of days in hospital.	Result.	Treatment	Remarks.
11 M. H.	F.	27	Mar.	7th	5	9	Father has had acute rheumatism	6	6	109·6°	Ankles, knees, shoulders, hands	Endocarditis	Previous	P.M. mitral and aortic cusps thickened and puckered, no recent endocarditis, heart 15 ozs., congestion of the lungs	6	Death	Hst. sod. sal., wet pack, antipyrin	She suffered from chorea on recovering from first attack of acute rheumatism. Death occurred from hyperpyrexia, which was uncontrolled by wet pack. Temp. 4 p.m...104° „ 10 „ ..107° „ 11 „ ..109·6°
12 E. A. A.	F.	33	„	6th	1	33	See previous admission No. 4	3	2	99·2°	Shoulders, elbows, hands	Endocarditis	„	Mitral regurgitation, apex in normal position	7	Recovery	Quinine antipyrin	Slight pain came on a week after discharge in February.
13 F. B.	M.	30	„	42nd	1	22	Father has had acute rheumatism 3 times, 1 brother suffers	2	3	101·2°	Right hand and foot	—	—	—	13	„	—	—

							from epilepsy										
14 J. McG.	M.	26	„	7th	3	12	Father died of D. T.	12	12	102·2°	Shoulders, knees	Pericarditis	Present	Heart sounds normal	24	„	Hst. sod. sal.	—
15 E. J.	F.	29	„	35th	1	23	Father and mother both suffered from acute rheumatism, mother died of consumption	3	2	102·4°	Ankles and feet	Endocrditis	?	Mitral regurgitation	14	„	„	Was in Middlesex Hospital until 10 days before admission with acute rheumatism, this attack being a relapse.
16 E. D.	F.	25	April	21st	0	25	—	11	13	102·0°	Knees, wrists, ankles, elbows	Pericarditis, systolic murmur at all orifices	Present	Heart sounds normal	32	„	„	Very prostrate on admission.
17 A. H.	M.	31	„	6th	2	27	Abrother suffers from rheumatism	35	20	105·4°	All large joints	Pericarditis, endocarditis	Present and previous	Mitral regurgitation	39	„	Hst. quinin.	A policeman. Came in with tonsillitis. Rheumatic arthritis came on 3 days afterwards; he became very prostrate, with lobar pneumonia, sickness, diarrhœa, and albuminuria; extensive desquamation, which was thought to be scarlatinal, followed.
18 A. P.	F.	27	„	9th	0	27	Father and a sister have had acute rheumatism, mother and a brother have heart disease	5	4	102·0°	Knees, ankles, shoulders	Soft systolic murmur at apex	Present	Heart sounds normal	23	„	Hst. pot. cit.	

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart. Affection.	Heart. In which attack developed.	Heart. State on discharge.	No. of days in hospital.	Result.	Treatment	Remarks.
20 M. A. G.	F.	17	May	3rd	4	9	Father died of heart disease	5	4	102·4°	Ankles and knees	Pericarditis, endocarditis	Present and previous	Mitral regurgitation	15	Recovery	Hst. sod. sal.	Chorea on recovering from previous attack of acute rheumatism in hospital, and 3 times since.
21 E. J.	F.	29	"	7th	2	23	See previous admission, No. 15	8	13	103·0°	Ankles, left knee, wrists, and shoulders	Endocarditis	Previous	Mitral regurgitation	23	"	"	—
22 E. W.	F.	26	"	7th	1	23	Father and 2 sisters died of consumption	1	3	103·0°	Knees, ankles	—	—	Heart sounds normal	16	"	"	—
23 L. C.	F.	27	"	6th	0	27	Father and a sister died of consumption	3	4	102·0°	Knees, left ankle	Systolic murmur at apex and over pulmonary cartilage	Present	Systolic over pulmonary cartilage	12	"	"	Had had sore throat for 5 weeks before admission, was recovering from follicular tonsillitis on admission.
24 C. H. G.	M.	40	June	8th	0	40	Father (twice) and a sister have had acute	18	18	108·0°	Wrists, left big toe	Pericarditis	"	P.M. exam., slight pericarditis, no endo-	18	Death	Wet pack, ice enemata, ice to suck, brandy,	Went on well for 3 days after admission, only sweating profusely. Then temp. rose to 106·6°, brought down by pack, &c. Temp.

						rheumatism							carditis, œdema of lungs, numerous rod-shaped bacilli in blood			quinine, hst. sod. sal.	rose over 105° on 6 occasions, namely, to 108°, 106·8°, 106°, 105·8°, 105·6°, always controlled by wet packs, of which altogether 31 were applied, and 4 ice enemata given. Death finally occurred from exhaustion, produced by frequent hyperpyrexia. Unconscious for 3 days before death.
25 L. W.	F.	16	„	6th	6	1	—	20	28	103·2°	Knees, ankles	Pericarditis	„	Heart sounds normal	59	Recovery	Hst. sod. sal., hst. effervescens.	Severe case.
26 E. C.	F.	43	„	10th	0	43	A sister suffers from rheumatism, 2 sisters and maternal grandfather died of consumption	14	16	103·6°	Knees, ankles	Pericarditis, endocarditis	„	Mitral regurgitation	35	„	Hst. sod. sal., hst. pot. cit.	—
27 M. A. L.	F.	32	„	6 mos.	0	32	—	8	23	102·6°	Knees, ankles, hands	Endocarditis	Previous	Double mitral presystolic thrill	34	„	Hst. sod. sal., sul. phonal.	Had been laid up in bed since the beginning of the year with joint pain and sweating. Confined during this period. Unable to feed herself, and consequently much exhausted on admission.
28 J. S.	M.	22	July	2nd	0	22	Mother died of consumption, aunt has fits	4	14	100·0°	Ankles, knees	Endocarditis	„	Mitral regurgitation	23	„	Hst. pot. tod. alb.	—

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart. Affection.	Heart. In which attack developed.	Heart. State on discharge.	No. of days in hospital.	Result.	Treatment	Remarks
29 S. H.	M.	20	July	10th	4	14	—	*14	9	102·2°	Knees	Endocarditis (probably ulcerative)	Present	No P.M. allowed; for some weeks before death double aortic and double mitral murmurs were heard	153	Death	Hst. quinin., pil. opii.	* Temp. was raised during the whole time he was in hospital, long after all symptoms of acute rheumatism had gone. Fever hectic in type, normal in the morning, up to 101°—103° every evening. Although admitted as a case of acute rheumatism he very soon developed symptoms of chronic ulcerative endocarditis; probably splenic infarction took place in October. Intense headache some days before death with rapid heart failure.
30 H. K.	F.	32	„	21st	0	32	Father suffers from gout, and has been paralysed for years	21	18	102·4°	Knees, ankles, wrists, elbows	—	—	Heart sounds normal	32	Recovery	Ol. morrh., liq. arsenicalis, antipyrin	Right facial paralysis since 7 years old. Advanced phthisis right apex.
31 B. B.	F.	18	„	10th	0	18	Mother has consumption	7	8	104°	Right shoulder, knees	—	—	„	25	„	Hst. sod. sal.	Sleeping in damp sheets just previously. Very anæmic.
32 C. C.	F.	17	Aug.	49th	2	16	See previous admission, No. 3.	3	1	102·0°	Hips, knees, ankles	Endocarditis	Previous	Mitral regurgitation	25	„	Hst. sod. sal.	—

33 W. H.	M.	25	Sept.	3rd	0	25	Mother died from paralytic stroke, 2 paternal aunts died of dropsy	4	3	104·0°	Knees, ankles, feet	Pericarditis, endocarditis	Present	"	52	"	Hst. sod. sal., hst. quinin.	Twelve days before admission had tonsillitis, which began with a rigor.
34 F. R.	M.	21	"	5th	2	17	Father died of hæmoptysis, æt. 35	14	9	103·2°	Knees, ankles, hands, right shoulder	Endocarditis	Previous	Aortic and mitral regurgitation, apex beat outside nipple	47	"	Hst. sod. sal., hst. quin.	—
35 E. C.	F.	16	"	5th	0	16	1 brother has rheumatism, father died of cancer of stomach	6	10	102·6°	Knees, ankles	Soft systolic murmur over pulmonary cartilage	—	Soft systolic over pulmonary cartilage	12	"	Hst. ferri cit.	An actress, dancing on a cold wet stage.
36 G. A.	M.	45	"	14th	0	45	—	3	9	102·4°	Knees, left wrist, shoulders	Pericarditis	Present	Heart sounds normal	16	"	Hst. sod. sal.	—
37 H. G.	M.	28	"	14th	0	28	—	5	9	100·8°	Left knee	—	—	"	15	"	Hst. sod. sal., hst. ferri cit.	—
38 W.W.	M.	24	"	8th	8	12	—	2	3	103·0°	Knees, ankles, left hip	Pericarditis, endocarditis	Present and previous	Mitral regurgitation	7	"	Hst. sod. sal.	—

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart. Affection.	Heart. In which attack developed.	Heart. State on discharge.	No. of days in hospital.	Result.	Treatment	Remarks.
39 G. W.	F.	15	Sept.	3rd	1	10	Father suffers from rheumatism	4	13	101·8°	Knees, ankles, right wrist, left elbow	Systolic murmur at apex	?	Systolic murmur at apex, apex in normal position	21	Recovery	Hst. sod. sal.	—
40 W. J.	M.	18	"	4th	1	10	Mother was insane for 3 years, maternal aunt has had acute rheumatism, one cousin has fits	4	8	103°	Knees, ankles	Endocarditis	Previous	Mitral regurgitation, apex outside nipple	14	"	"	—
41 G. W.	M.	40	"	7th	1	30	Father had hemiplegia, paternal aunt had acute rheumatism	4	13	103°	Hips, knees, ankles, wrists, and left shoulder	Soft systolic murmur at apex	—	Heart sounds normal	26	"	Hst. sod. sal., hst. quinin.	—
42 M. W.	F.	28	"	4th	4	16	Father and mother	27	25	104·0°	All large joints	Pericarditis, endo-	Present and	Mitral regurgitation,	43	"	Hst. sod. sal, hst. pot. cit.	Had pericarditis in a previous attack of acute rheumatism. Soon after

							both died of consumption maternal aunt had acute rheumatism					carditis	previous	apex outside nipple			sulphonal	admission this time developed pneumonia, crisis on 7th day accompanied by severe diarrhœa.
43 A. K.	M.	18	Oct.	35th	0	18	—	4	6	103°	Knees, ankles, right wrist	—	—	Heart sounds normal	12	„	Hst. sod. sal.	—
44 J. C.	M.	35	„	2nd	0	35	Father, mother, 2 brothers, and a sister all died of consumption	1	8	102·6°	Knees, ankles	—	—	„	23	„	„	—
45 B. B.	M.	19	„	42nd	1	19	Father (twice) and 2 brothers have had acute rheumatism	30	14	103°	Fingers, ankles, elbows	Pericarditis, endocarditis	Present	P.M. heart much enlarged, extensive pericarditis, rough lymph, 10 ozs. of fluid, vegetations on aortic and mitral valves, double pleuritis	30	Death	Pulv. ipec. co., brandy, pot. bicarb.	Very prostrate with much pericarditis when first admitted, improved, relapsed on 11th day, fresh pericarditis on 17th day. Died from aortic regurgitation.

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart. Affection.	Heart. In which attack developed.	Heart. State on discharge.	No. of days in hospital.	Result.	Treatment	Remarks.
46 H. C.	M.	26	Oct.	42nd	1	19	—	3	4	100·8°	Knees, ankles, right wrist, and metacarpo-phalangeal joints	Endocarditis	Present	Faint mitral systolic murmur, apex in nipple line	30	Recovery	Pulv. ipec. co.	Pleural friction in both axillæ during attack.
47 W. M.	M.	15	Nov.	3rd	1	15	Father died insane, æt. 39, mother has fits, a sister has had acute rheumatism	9	4	101·6°	Knees, ankles, hips, wrists	Endocarditis	"	Mitral regurgitation, apex in normal position	25	"	"	Gets constantly wet at his work in blacklead manufactory. Sore throat 14 days before admission. Herpes on nose.
48 S. B.	F.	18	"	4th	1	12	2 maternal aunts and 2 uncles died of consumption	3	4	102·4°	Knees, ankles	Pericarditis, endocarditis	Previous	Mitral regurgitation, ? presystolic murmur, apex in nipple line	42	"	Hst. sod. sal.	
49 M. L.	F.	8	"	2nd	0	8	Father had acute rheuma-	20	11	101·8°	Fingers and toes	Pericarditis, systolic murmur	Present	Heart sounds normal	22	"	Hst. pot. cit.	Began with a rigor.

							tism, maternal auntdied of consumption					at apex					
50 W. P.	M.	29	„	10th	0	29	Father died æt. 44, of consumption a brother had slight rheumatism	40	40	103·2°	Right ankle and knee, elbows and shoulders	Pericarditis, endocarditis	„	Mitral regurgitation, double aortic murmur, apex in normal position	50	„	Hst sod. sal.	A severe and persistent case. Discharged in January, 1890.
51 A. M.	F.	20	„	4th	2	13	Mother died of consumption æt. 30	3	13	99·6°	Ankles, knees, elbows, wrists	Pericarditis, systolic murmur at apex and over pulmonary valve	„	Heart sounds normal	21	„	„	—
52 C. S.	M.	26	Dec.	7th	0	26	—	9	9	102·6°	Wrists, ankles, knees, swelling very slight	Pericarditis, endocarditis	„	P.M., extensive pericarditis, partially adherent, about 4 ozs. blood-stained fluid. One small vegetation on mitral valve. Double pleuritis	9	Death	Brandy	A policeman. Sore throat 15 days before admission. Pericarditis developed early, great prostration, sleepless. constant delirium, gradual failure.
53 A. H.	M.	37	„	28th	0	37	Mother chronic sciatica	5	6	101·0°	Knees, ankles, wrists, shoulders	—	—	—	12	Recovery	—	Sweated profusely.

Table D.—*Chorea.*

During the years 1881-2-3-4-5-6-7-8-9 178 cases of chorea have been treated in the Hospital. A summary of these cases is prefixed to the analysis of the cases of the year 1889.

Inheritance.

Rheumatism	62
Neurosis	56
Phthisis	17
Gout	5

Sex.

	Males.	Females.
1881	6	14
1882	4	18
1883	3	14
1884	11	17
1885	7	12
1886	5	11
1887	12	12
1888	6	13
1889	3	10
Total	57	121

Ages.

	Under 5.	5—10.	10—20.	20—30.	30—40.	40—50.	50—60.
1881	0	4	17	1	0	0	0
1882	0	4	13	2	1	0	0
1883	1	4	10	2	0	0	0
1884	0	8	17	2	0	0	1
1885	0	3	14	1	0	1	0
1886	0	6	8	2	0	0	0
1887	2	5	17	0	0	0	0
1888	0	5	14	0	0	0	0
1889	0	2	10	1	0	0	0
Total	3	41	120	11	1	1	1

Rheumatism.—There was a history of acute rheumatism in 51 cases. The relation of the acute rheumatism to the chorea was as follows:—In 15 cases the rheumatism was suggested as the cause; in 13 cases the attack of rheumatism has developed during the course of the chorea; in the remaining 23 cases

there was a distinct interval of good health between the rheumatism and the chorea, viz., 6 years (2), 5 years, 4 years, 3 years (2), 2 years, 1 year (3), 7 months, 6 months (2), 5 months, 4 months, 3 months (3), 2 months (2), 6 weeks, 1 month (2).

Causes.—In 109 cases of the whole the causes assigned by the friends were fright, sorrow, or overwork at school.

Heart.—In 29 cases a mitral murmur constant in force and rhythm was audible; of these 29 cases 16 had suffered acute rheumatism previously. In 77 cases a murmur was heard over the apex occasionally only, the murmur varying in time and intensity, and having quite disappeared at the discharge of the patient.

TABLE D—*continued.*

No. and Name.	Family history.	Previous illness, with special reference to rheumatism.	Previous chorea.	Sex.	Age.	Heart condition. In Hospital.	Heart condition. On Discharge.	Part of body affected.	Cause.	Result.	Number of Days in Hospital.	Remarks.
1 A. H.	A brother died æt. 19 of acute rheumatism and chorea, a sister died of heart disease and phthisis	—	1	F.	23	Action irregular, occasional systolic murmur at apex	Heart sounds normal, action regular	General, face especially, speech much affected	Frightened by her sister falling out of a window and being killed	Relief	13	Was married 2 years ago, 3 miscarriages since, bad throat just after first miscarriage, tertiary syphilitic rash on face at time of admission.
2 E. H.	—	—	0	F	14	Action irregular, systolic apex murmur, apex in normal position	Systolic apex murmur, action more regular	Right arm and leg, face and tongue, speech much affected	—	Relief	21	Saw a girl with chorea at school 4 months previously. Treated with arsenic.
3 F. C.	Father suffers from "rheumatism," paternal great grandfather died of acute rheumatism	Subject to colds	0	F.	10	Action regular, systolic murmur all over præcordia, apex in normal position systolic murmur was noticed a month before ad-	Loud systolic murmur, apex in nipple line	All limbs, face and tongue, speech much affected, movements gradually ceased followed by general paresis, she improved much	—	Relief	35	From being an intelligent girl, she became very emotional, and then quite weak-minded; she then gradually improved but still remained emotional.

						miss'on)		before discharge, but there was still great weakness of gait and grasp, and some facial paralysis				
4 S. G.	Father suffered from "rheumatic gout"	Slight rheumatic pains and joint swellings æt. 5	3	F.	11	Action irregular, occasional systolic murmur at apex, a ex 1 inch outside nipple in 5th space	Heart sounds normal, action regular	Right arm most, left arm, face, tongue, legs very slightly	—	Relief	42	The first attack of chorea came on about 12 months after the rheumatism, there being no cause known.
5 L. L.	—	—	0	F.	5	Heart sounds normal	Heart sounds normal	Both arms, both legs, face and tongue	Working hard for an examination	Relief	79	This child had an attack of scarlet fever while in hospital. The temp. rose to 103°. The movements were not influenced by the fever.
6 G. H.	—	—	2	M.	14	Loud rough systolic murmur at apex, conducted into axilla. Slight hypertrophy	Remains the same	General, slight	Possibly overworking himself	Relief	49	Sent out for a fortnight in the middle on account of the Wards being cleaned.
7 J. P.	A sister had acute rheumatism	Measles	0	F.	12	Action irregular, no murmur	Action regular. Heart sounds normal	General, right side most affected, movements excessive	Reading hard for an examination	Relief	34	—

TABLE D—*continued.*

No. and Name.	Family history.	Previous illness, with special reference to rheumatism.	Previous chorea.	Sex.	Age.	Heart condition. In Hospital.	Heart condition. On Discharge.	Part of body affected.	Cause.	Result.	Number of Days in Hospital.	Remarks.
8 A. B	Mother had chorea and hysteria when the boy was 12 months old	Measles	0	M.	9	Action regular, sounds normal, epigastric pulsation	Heart sounds normal	General	Over-working at school. He saw a man with paralysis agitans, whom he used to imitate	Relief	32	—
9 R. U.	A sister died of brain fever	—	0	F.	12½	Action irregular, heart sounds normal	Action regular, sounds normal	General, especially right side, speech much affected, followed by paresis of limbs and tongue, which was first affected	Working hard for an examination, and doing a good deal of house work as well	Relief	84	Given liq. arsenicalis from 4 up to 8 min. t. d. s. for 6 weeks, stopped at last on account of giddiness. No apparent effect. She got up while still moving very much, and rapidly improved. Movements almost ceased on discharge.
10 A. H.	—	Swelling and pain in right knee and elbow 6 weeks before admission	0	F.	11	Double mitral murmur and thrill, apex in 6th space outside nipple	Remains the same	Slight movements of right arm, which were followed by paresis. The choreic	—	Relief	55	She came in with swelling, redness, and pain in right knee and wrist. Temp. was raised to 102·4°, and continued febrile for 10 days.

								movements gradually became general				
11 A. L.	Mother suffers from rheumatism	For the past 2 years has had irregular movements of the limbs	1	M.	11	Action irregular, systolic murmur at apex, apex in normal position	Heart sounds normal	General, more in left hand, the face, and tongue. They afterwards became worse, and were followed by slight paresis	Working for an examination	Relief	55	He said that he was frightened while in hospital by a delirious patient getting out of bed.
12 J. G.	Father has had acute rheumatism, mother and a sister died of phthisis, 1 brother had fits as a child	Three months ago had pains in the joints measles always coughs, had convulsions as a child	0	F.	10	Action slightly irregular, sounds normal	Action irregular, sounds normal	General, especially face and tongue, more on the left side	—	Relief	36	—
13 A. R.	—	Diphtheria 3 years ago	0	F.	7½	Action slightly irregular, pulmonary systolic, occasional apex systolic murmur, apex in normal position	Rough apex systolic murmur, action irregular	General, tongue not affected, considerable control of movements	Was frightened by some dogs at school, of which she had often spoken to her mother,	Recovery	40	—
								she was also anxious about her school work				

TABLE E.—*List of Deaths in the Medical Wards*

	Males.		Females.
GENERAL DISEASES:			
Morbilli	2	...	0
Enteric fever	0	...	2
Diphtheria	4	...	4
Pyæmia	1	...	0
Varicella	1	...	0
Acute rheumatism	4	...	1
Tuberculosis	3	...	6
DISEASES OF THE NERVOUS SYSTEM:			
Brain hæmorrhage	0	...	1
Cerebritis	1	...	0
Cerebral Abscess	0	...	1
Cerebral Tumour	1	...	1
Hydrocephalus	2	...	0
Meningitis	5	...	0
Delirium Tremens	2	...	0
Infantile convulsions	1	...	0
DISEASES OF THE CIRCULATORY SYSTEM:			
Dilated Heart	3	...	0
Mitral disease	3	...	10
Aortic disease	2	...	1
Pericarditis	1	...	0
Aneurysm, thoracic	1	...	0
DISEASES OF THE THYROID GLAND:			
Malignant disease	0	...	1
DISEASES OF THE RESPIRATORY SYSTEM:			
Bronchitis, acute	1	...	0
" chronic	4	...	5
Pneumonia, lobar	11	...	6
" lobular	8	...	4
Pleural effusion	1	...	0
Empyema	0	...	1
Phthisis	8	...	3
Emphysema	0	...	1
DISEASES OF THE DIGESTIVE SYSTEM:			
Epithelioma of the fauces	1	...	0
Carcinoma of the Stomach	3	...	0
Diarrhœa	5	...	4
Constipation	0	...	1
Enteritis	1	...	0
Duodenal ulcer	1	...	0
Typhlitis	1	...	0
Perforation of vermiform appendix	1	...	0

	Males.		Females.
Diseases of the Digestive System—*continued.*			
Malignant disease of intestine	1	...	0
" " " pancreas	1	...	0
" " " liver	1	...	2
Hepatic abscess	0	...	1
Acute peritonitis	0	...	1
Splenic leucocythæmia	1	...	0
Diseases of the Urinary System:			
Chronic nephritis, large white kidney	0	...	1
" " granular contracted kidney	4	...	2
" " suppurative	0	...	1
Marasmus	1	...	1
Total	92	...	62

SURGICAL REGISTRAR'S REPORT FOR THE YEAR 1889.

By ALFRED E. TAYLOR.

The total number of cases under treatment in the surgical wards during the past year was 1084, viz.:

	Males.	Females.	Total.
Remaining in hospital December 31st, 1888	39	29	68
Admitted during the year 1889			1016
			1084
Of these there were :			
Discharged, cured, or relieved	556	366	922
" unrelieved	28	16	44
Transferred to the medical wards	7	6	13
Died	25	10	35
In hospital December 31st, 1889	37	33	70
			1084

The mortality during the year was 3 22 per cent.

TABLE I.—*Showing the total number of Cases of each Disease or Injury which were Discharged or Died during the year* 1889.

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889.	
		M.	F.	M.	F.	M.	F.	M.	F.
Erysipelas:									
i. Admitted:									
a. Cutaneous	2	...	1	...	...	...	...	1	...
b. Cellulo-cutaneous	1	...	...	...	...	...	1	...	...
ii. Arising in hospital:									
a. Cutaneous	3	3	...	...	...	...	...	...	...
Pyæmia	3	1	...	...	...	1	...	...	1
Rickets	4	3	1	...	...	...	...	...	...
SYPHILIS:									
Squamæ	1	...	1	...	...	...	...	...	...
Gummata	9	4	5	...	...	...	...	...	...
Ulcers	11	3	8	...	...	...	...	...	...
Disease of tongue	1	...	1	...	...	...	...	...	...
Chancre of lip	1	...	1	...	...	...	...	...	...
Condylomata	4	...	3	1	...	...	...	...	...
TUMOURS:									
Scirrhus:									
i. *Primary:*									
Breast	9	...	8	...	...	...	1	...	...
ii. *Secondary:*									
Breast	2	...	2	...	...	...	...	...	...
Epithelioma:									
Lip	2	2	...	...	...	...	...	...	...
Palate	1	...	...	1	...	...	...	...	...
Œsophagus	1	...	...	...	...	1	...	...	...
Rectum	7	...	2	3	1	1	...	...	...
Penis	2	...	...	1	...	1	...	...	...
Scrotum	1	1	...	...	...	...	...	...	...
Toe	1	...	1	...	...	...	...	...	...
Sarcoma:									
Multiple	1	...	...	...	1	...	...	...	...
Malar bone	1	...	1	...	...	...	...	...	...
Jaw	2	...	...	1	...	1	...	...	...
Femur	1	...	...	...	1	...	...	...	...
Septum nasi	1	...	1	...	...	...	...	...	...
Glioma of retina	1	1	...	...	...	...	...	...	...
Skin	2	1	1	...	...	...	...	...	...
Myxoma:									
Nose	3	1	2	...	...	...	...	...	...
Rectum	1	...	...	...	...	...	...	1	...
Lipoma:									
Abdominal wall	1	...	1	...	...	...	...	...	...
Buttock	1	1	...	...	...	...	...	...	...
Angeioma:									
Nævus	3	2	1	...	...	...	...	...	...
Adeno-fibroma:									
Breast (cystic)	1	...	...	...	1	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889.	
		M.	F.	M.	F.	M.	F.	M.	F.
TUMOURS—*continued.*									
Papilloma:									
Chin	1	1	...	...	...	...	...	...	...
Vulva	4	...	3	...	1	...	...	...	...
Cysts:									
i. *Sebaceous:*									
Forehead	2	2	...	...	...	...	...	...	...
Scalp	3	1	2	...	...	...	...	...	...
ii. *Fibrous:*									
Submental	1	1	...	...	...	...	...	...	...
Ganglion	1	1	...	...	...	...	...	...	...
MALFORMATIONS:									
Cleft palate	1	...	1	...	...	...	...	...	...
Spina bifida	1	...	1	...	...	...	...	...	...
Imperforate anus	1	1	...	...	...	...	...	...	...
Genu valgum	5	2	3	...	...	...	...	...	...
Talipes varus	4	2	1	...	...	...	...	...	1
„ equinus	1	...	1	...	...	...	...	...	...
„ equino-varus	6	1	3	1	...	...	...	1	...
„ planus	6	4	1	...	1	...	...	...	...
Hammer-finger	1	...	1	...	...	...	...	...	...
DISEASES OF THE NERVOUS SYSTEM:									
Meningitis	1	...	...	...	...	...	1	...	...
Charcot's disease	1	1	...	...	...	...	...	...	...
Epilepsy	2	2	...	...	...	...	...	...	...
Hysteria	1	...	1	...	...	...	...	...	...
Infantile paralysis	3	1	2	...	...	...	...	...	...
Sciatica	2	...	1	...	...	...	...	1	...
Delirium tremens	4	1	...	...	...	3	...	...	...
Cerebral hæmorrhage	1	1	...	...	...	...	...	...	...
„ abscess	1	...	...	...	...	...	1	...	...
„ tumour	1	...	...	1	...	...	...	...	...
Insular sclerosis	1	...	...	1	...	...	...	...	...
DISEASES OF THE EAR:									
Purulent otitis media	11	4	3	...	...	...	3	1	...
Mastoid abscess	5	...	2	1	...	...	1	1	...
Polypus	1	1	...	...	...	...	...	...	...
DISEASES of THE EYE:									
Conjunctivitis	6	5	1	...	...	...	...	...	...
Conical cornea	1	...	1	...	...	...	...	...	...
Keratitis	2	1	1	...	...	...	...	...	...
Iritis	1	1	...	...	...	...	...	...	...
Kerato-iritis	2	2	...	...	...	...	...	...	...
Corneal ulcer	4	2	2	...	...	...	...	...	...
Cataract	8	4	3	...	...	...	...	1	...
Optic neuritis	1	...	...	1	...	...	...	...	...
Sympathetic ophthalmia	1	...	1	...	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF APPENDAGES OF EYE AND ORBIT:									
Ptosis	3	1	2	...	...	...	...	...	...
Strabismus	2	...	2	...	...	...	...	...	...
Ectropion	1	...	...	...	1	...	...	...	...
Obstructed lachrymal ducts	1	...	1	...	...	...	...	...	...
DISEASES OF THE NOSE:									
Polypus	3	1	2	...	...	...	...	...	...
Adenoid vegetations	6	3	3	...	...	...	...	...	...
Deviated septum	2	2	...	...	...	...	...	...	...
DISEASES OF THE CIRCULATORY SYSTEM:									
Thrombosis and phlebitis	2	1	1	...	...	...	...	...	...
Varicose veins	8	4	2	1	...	...	...	...	1
Aneurysm	6	2	...	2	...	...	...	2	...
Hæmatoma	11	6	5	...	...	...	...	...	...
Epistaxis	1	...	1	...	...	...	...	...	...
Hæmorrhage after extraction of tooth	1	1	...	...	...	...	...	...	...
DISEASES OF THE RESPIRATORY SYSTEM:									
Phthisis	1	...	1	...	...	...	...	...	...
Broncho-pneumonia	1	...	1	...	...	...	...	...	...
Acute bronchitis	1	...	1	...	...	...	...	...	...
DISEASES OF THE ABSORBENT SYSTEM:									
Glands:									
i. Inflammation	21	12	7	...	...	...	...	...	2
ii. Suppuration	34	18	14	...	...	...	...	...	2
Lymphangitis	22	16	3	...	...	...	...	1	2
Elephantiasis	1	...	1	...	...	...	...	...	...
DISEASES OF THE DIGESTIVE SYSTEM:									
Stomatitis	1	...	1	...	...	...	...	...	...
Hypertrophied tonsils	5	...	5	...	...	...	...	...	...
Œsophagus:									
Stricture of	2	1	...	...	...	1	...	...	...
Intestines:									
Peritonitis	4	3	...	...	...	...	1	...	...
Fæcal fistula	1	...	...	1	...	...	...	...	...
Hernia:									
a. Reducible	4	2	...	1	...	1	...	...	...
b. Irreducible	3	3	...	...	...	...	...	...	...
c. Incarcerated	2	1	1	...	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE DIGESTIVE SYSTEM—*continued.*									
Intestines:									
Hernia:									
d. Strangulated:									
i. Inguinal	18	13	1	...	...	4	...	...	...
ii. Femoral	2	1	...	...	...	...	...	...	1
iii. Umbilical	2	...	1	...	...	1	...	...	...
Rectum and Anus:									
Carcinoma	7	...	2	3	1	1	...	...	...
Stricture (fibrous)	3	...	2	...	1	...	...	...	...
Fistula	14	10	3	...	1	...	...	...	...
Hæmorrhoids	21	8	9	1	1	...	1	...	1
Fissure	4	3	1	...	...	...	...	...	...
Prolapsus	5	3	1	1	...	...	...	...	...
Ulcer	2	2	...	...	...	...	...	...	...
Polypus	1	...	...	...	...	...	...	1	...
DISEASES OF THE URINARY ORGANS:									
Kidney:									
Calculus	2	1	...	...	1	...	...	...	...
Bladder:									
Cystitis	5	3	1	...	...	...	1	...	...
Incontinence	2	1	...	...	1	...	...	...	...
Prostate:									
Hypertrophy of	6	5	...	...	...	...	...	1	...
Tubercular	1	...	...	1	...	...	...	...	...
Urethra:									
Ruptured urethra	1	1	...	...	...	...	...	...	...
Gonorrhœa and its Complications:									
Gonorrhœa and balanitis	16	10	5	...	...	...	...	1	...
Bubo	16	8	5	...	...	...	...	2	1
Stricture of urethra	21	15	...	...	...	3	...	3	...
Epididymitis	6	5	...	...	...	...	...	1	...
Orchitis	1	1	...	...	...	...	...	...	...
Perineal fistula	4	2	...	1	...	1	...	...	...
Prostatic abscess	1	...	...	...	...	...	...	1	...
Extravasation of urine	2	...	...	...	...	2	...	...	...
DISEASES OF THE GENERATIVE SYSTEM:									
MALE:									
Penis:									
Chancroid	14	12	...	...	...	...	...	2	...
Gangrene	1	...	...	...	...	1	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE GENERATIVE SYSTEM—*continued.*									
MALE:									
Penis:									
Phimosis:									
a. Congenital	28	28	...	...	...	...	...	...	...
b. Acquired	16	15	...	...	...	...	...	1	...
Paraphimosis	2	2	...	...	...	...	...	...	...
Tunica vaginalis:									
Hydrocele	3	3	...	...	...	...	...	...	...
Hæmatocele	1	1	...	...	...	...	...	...	...
Testis:									
Undescended	1	1	...	...	...	...	...	...	...
Tubercular	2	2	...	...	...	...	...	...	...
Cord:									
Varicocele	6	6	...	...	...	...	...	...	...
Hydrocele	1	1	...	...	...	...	...	...	...
FEMALE:									
Labium:									
Abscess	1	...	1	...	...	...	...	...	...
Vagina:									
Vaginitis	8	...	7	...	...	...	...	...	1
Fistula	2	...	...	...	1	...	...	...	1
Urethra:									
Caruncle	1	..	1	...	...	...	...	...	...
Breast:									
Inflammation	1	...	1	...	...	...	...	...	...
Abscess	7	...	7	...	...	...	...	...	...
Schirrhus:									
i. Primary	9	...	8	...	...	...	1	...	...
ii. Secondary	2	...	2	...	...	...	...	...	...
Adenoma	1	...	...	...	1	...	...	...	...
DISEASES OF THE BONES:									
Periostitis and ostitis:									
i. Acute:									
Femur	3	1	2	...	...	...	...	...	...
Tibia	4	3	...	...	...	1	...	...	...
Jaw	1	1	...	...	...	...	...	...	...
ii. Chronic:									
Femur	2	2	...	...	...	...	...	...	...
Tibia	2	1	1	...	...	...	...	...	...
Metacarpus	2	...	2	...	...	...	...	...	...
Metatarsus	2	1	...	...	...	...	...	...	1
Exostosis:									
Multiple	1	...	...	...	1	...	...	...	...
Great toe	1	1	...	...	...	...	...	...	...
Necrosis:									
Upper jaw	1	...	1	...	...	...	...	...	..
Lower jaw	3	2	...	...	...	...	...	1	...
Nasal bones	1	...	1	...	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF BONES—*continued.*									
Necrosis:									
Humerus	1	...	1	...	...	...	...	...	...
Phalanges	5	1	4	...	...	...	...	...	...
Caries:									
Spine	23	6	13	2	...	...	...	1	1
Tarsus	5	2	1	...	1	...	...	...	1
Carpus	1	1	...	...	...	...	...	...	...
Ilium	2	...	2	...	...	...	...	...	...
Ribs	3	3	...	...	...	...	...	...	...
Tibia (head of)	1	...	...	...	...	1	...	...	...
Metatarsus	3	1	1	...	...	...	...	...	1
DISEASES OF THE JOINTS:									
Synovitis:									
i. Acute:									
Ankle	4	2	2	...	...	...	...	...	...
Knee	14	10	4	...	...	...	...	...	...
Elbow	2	1	1	...	...	...	...	...	...
ii. Chronic:									
Knee	11	4	4	1	...	...	1	1	...
Ankle	1	...	1	...	...	...	...	...	...
Finger	1	1	...	...	...	...	...	...	...
Morbus coxæ	37	13	18	...	2	1	1	2	...
Arthritis:									
i. Acute:									
Knee	5	2	...	...	...	1	...	...	2
Shoulder	2	2	...	...	...	...	...	...	...
Sacro-iliac	1	...	...	...	1	...	...	...	...
ii. Strumous:									
Elbow	4	1	2	...	...	...	...	...	1
Knee	7	1	5	...	1	...	...	...	...
Ankle	5	3	1	...	...	...	...	...	1
Wrist	2	...	1	...	...	...	...	1	...
Tarsus	2	1	1	...	...	...	...	...	...
Chronic rheumatic arthritis	7	3	4	...	...	...	...	...	...
Charcot's disease of shoulder joint	1	1	...	...	...	...	...	...	...
"Flail" knee joint after excision	1	...	...	...	...	...	...	...	1
Ankylosis:									
i. Fibrous:									
Shoulder	3	1	1	1	...	...	...	...	...
Hip	1	1	...	...	...	...	...	...	...
Knee	1	...	1	...	...	...	...	...	...
Elbow	1	...	1	...	...	...	...	...	...
Foot	1	...	1	...	...	...	...	...	...
ii. Bony:									
Hip	1	...	1	...	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF MUSCLES, FASCIÆ, AND TENDONS :									
Spasmodic torticollis	1	1	...	...	...	...	...	...	...
Lateral curvature	13	1	12	...	...	...	...	...	...
Pulpy synovitis, sheath of tendons	1	...	1	...	...	...	...	...	...
Hysterical contraction of fingers	1	...	1	...	...	...	...	...	...
Lumbago	2	2	...	...	...	...	...	...	...
Bursæ :									
Submental cyst	1	1	...	...	...	...	...	...	...
Ganglion	1	1	...	...	...	...	...	...	...
Inflammation :									
i. Acute :									
Patella	14	6	5	...	1	...	...	...	2
Olecranon	3	3	...	...	...	...	...	...	...
Over head of fibula	1	1	...	...	...	...	...	...	...
ii. Chronic :									
Patella	4	...	4	...	...	...	...	...	...
Bunion	1	1	...	...	...	...	...	...	...
DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUE :									
Cellulitis :									
Face	1	1	...	...	...	...	...	...	...
Upper limb	17	12	4	...	...	...	...	1	...
Lower limb	4	1	3	...	...	...	...	...	...
Abscess :									
i. Acute :									
Scalp	4	2	1	...	...	...	...	1	...
Face	3	...	2	...	...	...	...	1	...
Jaw	5	1	3	...	...	...	...	1	...
Chest wall	1	1	...	...	...	...	...	...	...
Axilla	5	5	...	...	...	...	...	...	...
Hand	6	3	2	...	...	...	...	...	1
Ischio-rectal	9	8	1	...	...	...	...	...	...
Buttock	1	1	...	...	...	...	...	...	...
Perinæal	1	1	...	...	...	...	...	...	...
Leg	5	4	1	...	...	...	...	...	...
Foot	1	1	...	...	...	...	...	...	...
ii. Chronic :									
Neck	19	11	8	...	...	...	...	...	...
Chest	2	1	1	...	...	...	...	...	...
Abdomen	1	...	1	...	...	...	...	...	...
Thigh	1	...	1	...	...	...	...	...	...
Leg	1	1	...	...	...	...	...	...	...
Foot	1	1	...	...	...	...	...	...	...
Axilla	1	...	1	...	...	...	...	...	...
Arm	3	1	2	...	...	...	...	...	...
Loin	4	4	...	...	...	...	...	...	...
Iliac	1	1	...	...	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUE—*continued.*									
Gangrene:									
i. Moist	2	...	...	1	...	1	...	...	...
ii. Dry	3	2	1	...	...	...	...	...	...
Sinuses:									
Neck	2	2	...	...	...	...	...	...	...
Chest	1	1	...	...	...	...	...	...	...
Loin	1	...	...	...	1	...	...	...	...
Thigh	1	...	1	...	...	...	...	...	...
Foot	2	2	...	...	...	...	...	...	...
Great toe	1	...	...	...	...	...	...	...	1
Ulcers:									
i. Simple:									
Leg	8	3	5	...	...	...	...	...	...
Ankle	1	1	...	...	...	...	...	...	...
Foot	5	4	1	...	...	...	...	...	...
ii. Varicose:									
Leg	7	5	1	...	...	...	...	...	1
iii. Strumous:									
Tongue	1	...	...	...	...	...	...	1	...
Leg	1	1	...	...	..	...	...	...	...
iv. Traumatic:									
Foot	1	...	1	...	...	...	...	...	...
Leg	1	1	...	...	...	...	...	...	...
Carbuncle	6	4	...	...	...	...	...	1	1
Eczema	3	1	2	...	...	...	...	...	...
Scabies	3	1	2	...	...	...	...	...	...
Lupus	11	5	6	...	...	...	...	...	...
Rodent ulcer	1	...	1	...	...	...	...	...	...
Pemphigus	1	1	...	...	...	...	...	...	...
Ingrowing toe-nail	6	3	3	...	...	...	...	...	..
Suppurating corn	4	2	2	...	...	...	...	...	...
Erythema nodosum	1	...	1	...	...	...	...	...	...
UNCLASSIFIED:									
Painful stump	2	2	...	...	...	...	...	...	...
Malingering	1	1	...	...	...	...	...	...	...
Bronchocele	2	...	2	...	...	...	...	...	...
Needle in knee	2	...	2	...	...	...	...	...	...
Swallowed a pin	1	1	...	...	...	...	...	...	...
Contracted neck after burn	1	...	1	...	...	...	...	...	...
" fingers after burn	1	1	...	...	...	...	...	...	...
INJURIES:									
Burns:									
Local	8	2	4	...	...	...	...	...	2

TABLE I.—*continued.*

INJURIES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889.	
		M.	F.	M.	F.	M.	F.	M.	F.
INJURIES—*continued.*									
Scalds:									
i. General	2	...	1	...	...	1	...	...	...
ii. Local	12	5	4	...	...	1	...	1	1
Contusions:									
General	2	1	1	...	...	...	...	...	...
Immersion:									
Attempted suicide	7	1	6	..	...	..	...	...	...
Poisoning:									
Accidental	2	...	2	...	...	...	...	...	...
Injuries of head:									
Scalp wounds	28	19	7	...	...	...	...	...	2
Fractures (base)	3	1	...	...	...	2	...	...	...
Concussion of brain	14	9	4	...	...	1	...	...	...
Injuries of face:									
Contusion	6	3	1	...	...	1	...	...	1
Wounds (contused)	7	5	2	...	...	...	...	...	...
„ (incised)	3	3	...	...	...	...	...	...	...
„ (lacerated)	2	2	...	...	...	...	...	...	..
Fractured jaw	3	3	...	...	...	...	...	...	...
Injuries of eye:									
Wound of eyelid	5	5	...	...	...	...	...	...	...
„ cornea (punctured)	3	2	...	...	...	...	...	...	1
Ruptured eyeball	2	1	...	...	...	...	...	1	...
Injuries of neck:									
Cut throat (suicide)	2	1	...	...	...	1	...	...	...
Injuries of back:									
Fractured spine	1	...	...	1	...	...	...	...	...
Injuries of chest:									
Wound	2	2	...	...	...	...	...	...	...
Fractured ribs	5	5	...	...	...	...	...	...	...
Injuries of abdomen:									
Contusions	4	1	2	...	...	...	1	...	...
Injuries of pelvis and genitals:									
Punctured wound of testis	1	1	...	...	...	...	...	...	...
Injuries of upper extremity:									
Contusions	4	1	3	...	...	...	...	...	...
Wounds (contused)	2	...	2	...	...	...	...	...	...
„ (incised)	5	1	4	...	...	...	...	...	...
„ (lacerated)	2	2	...	...	...	...	...	...	...

TABLE I.—*continued.*

INJURIES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1889.	
		M.	F.	M.	F.	M.	F.	M.	F.
INJURIES—*continued.*									
Injuries of upper extremity:									
Crushed fingers	11	10	1	...	...	...	...	...	...
Fractures:									
Colles'		...	1	...	...	...	...	...	...
Clavicle	3	2	1	...	...	...	...	...	...
Humerus	9	4	3	...	...	1	...	1	...
Radius	2	...	1	...	...	...	...	...	1
Ulna	1	...	...	...	...	...	...	...	1
Metacarpus	1	1	...	...	...	...	...	...	...
Injuries of lower extremity:									
Contusions	12	6	6	...	...	...	...	...	...
Wounds (contused)	4	3	...	...	...	...	1	...	...
" (incised)	3	3	...	...	...	...	...	...	...
" (punctured)	1	1	...	...	...	...	...	...	...
Sprains:									
Ankle	5	2	2	...	...	1	...	...	...
Crushed feet	5	5	...	...	...	...	...	...	...
Fractures (simple):									
Femur (intracapsular)	2	...	2	...	...	...	...	...	...
" (extracapsular)	1	1	...	...	...	...	...	...	...
" (shaft)	13	7	4	...	...	...	...	2	...
Patella	7	3	4	...	...	...	...	...	...
Tibia	11	11	...	...	...	...	...	...	...
Fibula	2	2	...	...	...	...	...	...	...
Tibia and fibula	8	5	2	...	...	...	...	1	...
Potts'	10	8	2	...	...	...	...	...	...
Phalanx	1	...	1	...	...	...	...	...	...
Fractures (compound):									
Tibia	1	1	...	...	...	...	...	...	...
Tibia and fibula	3	2	1	...	...	...	...	...	...
Dislocations:									
Shoulder	2	...	2	...	...	...	...	...	...
Knee	1	1	...	...	...	...	...	...	...
Patella	2	1	1	...	...	...	...	...	...
Cervical vertebra	1	1	...	...	...	...	...	...	...
"Run over"—Shock	13	8	3	...	...	...	1	1	...
Starvation	1	1	...	...	...	...	...	...	...

TABLE II.—*Operations.*

OPERATIONS.	Total.	Discharged.		Died.		In hosp., 31st Dec., 1889.		Remarks.
		M.	F.	M.	F.	M.	F.	
HEAD AND NECK:								
For epithelioma of lip	2	2	...	...	...	...	...	
„ sarcoma of skin of face.	1	...	1	...	...	...	...	
„ periosteal sarcoma of jaw	1	...	...	1	...	...	...	Excision of half lower jaw.
„ periosteal sarcoma of malar bone	1	...	1	...	...	...	...	Excision.
„ naso-pharyngeal polypus	1	...	1	...	...	...	...	Lawrence's operation.
„ bronchocele	2	...	1	...	1*	...	...	*Extirpation.
„ venous nævus of neck	1	...	1	...	...	...	...	
„ adenoid vegetations in naso-pharynx	7	4	3	...	...	...	...	Curetting.
„ cleft palate	1	...	1	...	...	...	...	Staphylorraphy.
„ carbuncle of neck	3	3	...	...	...	...	...	Incision and "scraping."
„ lupus of face	7	4	3	...	...	...	...	
„ cut throat (suicidal)	1	...	...	1	...	...	...	Sutures.
„ trephining	3	...	...	...	3	...	...	For cerebral abscess after purulent otitis media.
EYES:								
For cataract	8	4	3	...	...	1	...	
„ strabismus	2	...	2	...	...	...	...	For internal strabismus.
„ hypopyon	1	...	1	...	...	...	...	Paracentesis corneæ.
„ ptosis	1	1	...	...	...	...	...	
Excision of globe	3	...	3	...	...	...	...	(1) for glioma of retina (2) for perforating ulcer of cornea (3) for staphyloma of cornea.
EARS:								
For aural polypus	1	1	...	...	...	...	...	
THORACIC REGION:								
For adenoma of breast	2	...	2	...	...	...	...	Amputation.
„ schirrhus „	7	...	7	...	...	...	...	Amputation.
„ chronic induration of breast	1	...	1	...	...	...	...	Amputation.
„ carbuncle of back	1	1	...	...	...	...	...	Incision and scraping.
„ necrosis of rib	1	...	1	...	...	...	...	Sequestrotomy.
ABDOMINAL REGION:								
For radical cure of hernia	3	3	...	...	...	...	...	
„ strangutaled inguinal hernia	7	3	1	2	...	...	...	
„ strangulated umbilical hernia	1	...	...	1	...	...	...	
„ umbilical fæcal fistula	1	...	...	...	...	1	...	Attempted closure.
„ malignant disease of œsophagus	1	...	...	1	...	...	...	Gastrostomy.
„ lipoma of abdominal wall	1	...	1	...	...	...	...	Excision.
„ renal calculus	1	1	...	...	...	...	...	Exploration.
„ spinal caries	1	...	...	...	...	...	1	For psoas abscess.
Colotomy	1	...	1	...	...	...	...	For cancer of rectum.

TABLE II.—*continued.*

OPERATIONS.	Total.	Discharged.		Died.		In hosp., 31st Dec., 1889.		Remarks.
		M.	F.	M.	F.	M.	F.	
ABOUT RECTUM AND ANUS:								
For hæmorrhoids	12	4	7	...	1	...	...	(1) Ligature (1) Whitehead's operation, 10 excision and cautery.
„ fistula	16	12	4	...	...	...	...	
„ fissure	2	2	...	...	...	...	...	
„ stricture of rectum	1	...	1	...	...	...	...	Dilatation
„ polypus „	1	...	1	...	...	...	...	Excision.
„ imperforate anus	1	1	...	...	...	...	...	
„ epithelioma of anus	1	1	...	...	...	...	...	Palliative.
ON THE GENITO-URINARY ORGANS:								
For stricture of urethra:								
(i) External urethrotomy	3	1	...	2*	...	...	...	*After extravasation of urine.
(ii) Internal „	1	1	...	...	...	...	...	
(iii) Dilatation	12	12	...	...	...	...	...	
For phimosis	44	44	...	...	...	...	...	
„ varicocele	4	4	...	...	...	...	...	Excision.
„ venereal warts	5	...	5	...	...	...	...	
„ radical cure of hydrocele	2	2	...	...	...	...	...	
„ vesico-vaginal fistula	1	...	...	...	...	...	1	After vaginal cystotomy for calculus.
„ castration	2	2	...	...	...	...	...	For tubercular testis.
ON UPPER LIMB:								
For ganglion of wrist	2	2	...	...	...	...	...	Incision and removal of "melon seed" bodies.
„ necrosis of humerus	2	1	1	...	...	...	...	Sequestrotomy.
„ fibrous ankylosis of shoulder	2	1	1	...	...	...	...	Breaking down adhesions
„ fibrous ankylosis of elbow	1	...	1	...	...	...	...	„ „ „
„ dislocation of shoulder	1	...	1	...	...	...	...	
„ pulpy disease of carpus	1	1	...	...	...	...	...	Arthrectomy.
„ „ „ elbow	2	1	1	...	...	...	...	Excision.
Suturing flexor tendons at wrist	1	1	...	...	...	...	...	
ON LOWER LIMB:—								
For sarcoma of skin of leg	1	1	...	...	...	...	...	Excision.
„ „ femur	1	...	1	...	...	...	...	Exploration.
„ exostosis of fibula	1	1	...	...	...	...	...	Removal.
„ „ great toe	1	1	...	...	...	...	...	„
„ nævus of leg	1	1	...	...	...	...	...	Ligature.
„ popliteal aneurism	3	1	...	...	...	2*	...	(*1 Diffuse), ligature of superficial femoral artery.
„ varix of leg	5	4	...	...	...	...	1	2 ligature, 3 excision.
„ sciatica	1	...	1	...	...	...	...	Stretching of sciatic nerve.

TABLE II.—*continued.*

OPERATIONS.	Total.	Discharged.		Died.		In hosp., 31st Dec., 1889.		Remarks.
		M.	F.	M.	F.	M.	F.	
ON LOWER LIMB—*cont.*								
For genu valgum	4	1	3	...	...	...	...	3 Ogston's, 1 MacEwen's operation.
„ talipes varus	4	1	2	...	...	...	1	3 excision of tarsal arch.
„ „ planus	5	3	2	...	...	...	...	Cautery.
„ „ equino-varus	1	...	1	...	...	...	...	Tenotomy.
„ chronic periostitis of femur	1	1	...	...	...	...	...	Incision.
„ necrosis of femur	2	1	1	...	...	...	...	Sequestrotomy.
„ „ tibia	1	1	...	...	...	...	...	„
„ „ metatarsus	1	1	...	...	...	...	...	Excision of bone.
„ caries of os calcis	3	1	1	...	...	...	1	Scraping.
„ ecurvation of tibiæ (rickets)	1	...	1	...	...	...	...	Osteotomy.
„ contracted hip	3	...	3	...	...	...	...	2 breaking down adhesions, 1 osteotomy of femur for faulty ankylosis.
„ „ knee	2	1	...	...	...	...	1	Breaking down adhesions.
„ chronic synovitis of knee	2	...	1	...	1*	...	..	Opening and draining. *From peritonitis.
„ suppurative arthritis of knee	1	...	...	...	...	...	1	Incision.
„ pulpy disease of knee	1	...	1	...	...	...	...	Arthrectomy.
„ „ „ ankle	1	...	1	...	...	...	...	„
„ morbus coxæ	8	5	2	1	...	...	...	6 incision and drainage, 1 trephining, 1 excision.
„ sacro-iliac disease	1	1	...	...	...	...	...	Exploration.
„ housemaid's knee	4	2	2	...	...	...	...	
„ ingrowing toe-nail	3	2	1	...	...	...	...	Avulsion.
„ needle in knee	1	...	1	...	...	...	...	
„ painful stump	1	1	...	...	...	...	...	Excision of bulbous nerve.
AMPUTATIONS :—								
Of fingers	17	12	5	...	...	...	...	
„ toes	6	4	2	...	...	...	...	
Through arm	1	1	...	...	...	...	...	For extensive suppurative cellulitis of forearm.
Syme's amputation	1	1	...	...	...	...	...	For suppuration in sole of foot.
Through leg	2	1	1	...	...	...	...	1 for crushed leg, 1 for "pulpy" ankle.
„ knee	2	1	...	...	...	1	...	1 for necrosis of tibia, 1 for gangrene of leg and foot after ligature of femoral artery for popliteal aneurism.
„ thigh	4	3	1	...	...	...	...	1 for crushed leg, 1 for necrosis of femur, 2 for pyæmic knee-joints.

TABLE III.—*Cases of Infectious Disease during the year* 1889.

Sex.	Age.	Date of admission.	Disease for which admitted.	Hospital disease.	Result and date.
M.	5 months	June 27th	Suppurative arthritis of knee joint	Pyæmia	Died July 14th.
F.	72	December 3rd ...	Contused wound of leg	Erysipelas	„ December 5th.
M.	36	March 11th	Suppurative arthritis of knee joint	Pyæmia	Relieved May 12th.
F.	16	April 9th	Pulpy disease of ankle—sinuses	Erysipelas	„ „ 25th.
F.	4	March 20th	Erysipelas of leg	„	„ March 24th.
M.	12	November 20th..	Scalp wound—Erysipelas	„	„ December 5th.
F.	62	December 10th ..	„ „ „	„	„ „ 16th.
F.	34	December 9th ...	Suppurative arthritis of knee joint	Pyæmia	„ January 24th, 1890.
M.	14	February 19th...	Popliteal abscess opening into knee.....	Erysipelas	„ May 10th.
M.	45	October 4th	Diffuse popliteal aneurysm, ligature of femoral, gangrene	„	„ January 24th, 1890.
M.	54	„	Abscess of foot	„	„ December 10th.
M.	8	November 4th...	Adenoid vegetations in naso-pharynx	„	„ November 12th.

REPORT OF CASES IN "ARDEN WARD"

(FOR DISEASES OF WOMEN)

DURING THE YEAR 1889.

By E. DAWSON.

Number of patients under treatment, January 1st		8
" " admitted during 1889		93
Total		101
Number of patients discharged during the year:		
Cured or relieved	78	
Unrelieved	5	
Transferred to other wards	2	
	—	85
*Died		5
Under treatment, December 31st, 1889		11
Total		101

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* 1 pelvic cellulitis, 1 puerperal eclampsia, 2 hysterectomy, 1 solid ovarian tumour after operation.

Cases in "Arden Ward."

Nature of Case.	No. of Case.	Initials.	Age.	Result.	No. in Register.	Remarks, treatment, &c.
Uterus:						
Subinvolution	I	S. P.	37	Relieved	M. 180	After parturition.
Retroflexion	I	S. J.	35	"	M. 73	
	II	F. C.	27	"	M. 75	
	III	M. S.	48	Unrelieved	M. 88	Chronic peri and parametritis.
	IV	L. L.	24	Relieved	M. 121	
	V	E. R.	41	"	M. 129	
	VI	A. P.	24	"	N. 39	
	VII	A. L.	39	"	N. 45	
	VIII	T. D.	30	"	N. 64	
Prolapse	I	E. G.	22	"	M. 91	
	II	J. W.	37	"	M. 187	Third degree. Death from phthisis one month after discharge
	III	C. T.	30	"	M. 232	First degree.
	IV	S. J.	50	"	N. 101	Third degree.
Fibroid	I	E. P.	41	"	M. 47	Interstitial. Electrolysis, 28 applications of ten minutes each.
	II	M. C.	49	"	M. 55	Subperitoneal. Incarcerated. Elevated.
	III	C. M.	30	Cured	M. 69	Submucous. Sloughing; removed.
	IV	S. W.	47	Death	M. 80	Interstitial. Hysterectomy. Pyosalpinx, and scirrhus of pylorus.
	V	E. H.	39	Unrelieved	M. 106	Interstitial. Transferred to an Infirmary.
	VI	E. A.	52	Cured	M. 117	Submucous. Removed with scissors.
	VII	L. T.	46	Death	M. 171	Subperitoneal. Abdominal section, removal. Death from shock.
	VIII	L. G.	39	Cured	M. 214	Submucous. Pedunculated. Removed by ecraseur.
	IX	D. G.	47	"	N. 18	Submucous. Pedunculated. Removed by scissors, cautery to base.
	X	E. M.	46	Relieved	N. 26	Interstitial. Electrolysis, 30 applications of ten minutes each.
	XI	M. F.	35	Unrelieved	N. 53	Interstitial. Electrolysis, 6 applications of ten minutes each. Perityphlitis.
	XII	L. L.	40	Relieved	N. 55	Interstitial. Electrolysis, 14 applications of ten minutes each.
Polypus	I	L. S.	42	Cured	M. 107	Mucous, removed with scissors.

	II	K. B.	37	"	M. 153	Removed, with wire ecraseur.
	III	R. D.	56	"	M. 159	Mucous, removed by torsion.
	IV	A. N.	23	"	M. 161	Mucous, removed by torsion.
Malignant Disease	I	M.H.T.	45	Relieved	M. 67	Sarcoma of body.
	II	J. S.	40	"	M. 109	Encephaloid carcinoma. Removed as far as possible.
	III	C. E.	35	"	M. 112	Epithelioma of cervix. Removed with scissors, and actual cautery. Abortion of a four months' fœtus.
	IV	L. D.	46	"	M. 119	Epithelioma of cervix. Removed. Cautery to base.
	V	M. A. F.	32	"	M. 125	Epithelioma of cervix. Sharp spoon and cautery.
	VI	E. W.	30	Unrelieved	M. 137	Carcinoma of cervix. Too advanced for operation.
	VII	A. C.	67	Relieved	M. 166	Epithelioma of cervix.
	VIII	G. W.	70	Unrelieved	M. 225	Carcinoma. Rectum implicated. Too advanced for operation. Sent to Infirmary.
	IX	E. B.	30	Relieved	N. 4	Epithelioma of cervix.
	X	G. W.	32	"	N. 70	Carcinoma. Operation refused.
Cervix Uteri:						
Stenosis	I	E. H.	31	Cured	M. 71	Never pregnant. Division of cervix with metrotome, relief of dysmenorrheal pains.
	II	M.A.H.	22	"	N. 47	Never pregnant. Division of cervix with metrotome, relief of dysmenorrheal pains.
Endocervicitis	I	L. D.	24	Relieved	M. 219	Chromic acid used.
	II	L. H.	23	"	M. 228	
Syphilitic Ulceration ...	I	A. H.	43	"	M. 192	
	II	E. D.	28	"	N. 59	Improved under specific treatment.
	III	E. M.	37	"	N. 61	Improved under specific treatment.
Ovary:						
Ovaritis	I	C. G.	43	"	M. 63	Chronic.
	II	A. H.	40	"	N. 8	Chronic.
	III	E. B.	28	"	N. 43	Chronic.
Tumours—						
Solid	I	J. S.	32	Death	M. 59	Ovariotomy. Hæmorrhage from pedicle. Weight of tumour nearly 9 lbs.
Cystic	I	S. C.	66	Cured	M. 96	Ovariotomy.
	II	F. A.	28	"	N. 35	Ovariotomy.

CASES IN "ARDEN WARD"—*continued.*

Nature of Case.	No. of Case.	Initials.	Age.	Result.	No. in Register.	Remarks, treatment, &c.
VAGINA:						
Abscess of Bartholin Gland	I	A. R.	23	Cured	M. 196	Right gland. Opened and plugged.
	II	M. B.	24	"	M. 198	Right gland. Opened and plugged.
Hæmatoma of Labium	I	S. H.	35	"	M. 211	Traumatic. Ruptured naturally. Cavity plugged.
BLADDER, URETHRA, &c.:						
Cystitis	I	D. S.	60	Relieved	M. 156	Subacute.
	II	S. H.	33	"	M. 208	Chronic.
Incontinence of Urine	I	A. A.	30	"	M. 133	Bladder contracted.
Caruncle	I	S. B.	31	Cured	M. 100	Scalpel and Paquelin's cautery.
	II	J. N.	46	Relieved	M. 150	Scalpel and Paquelin. Recurred after removal.
	III	M. A. S.	67	"	M. 206	Scalpel and actual cautery.
	IV	F. M.	27	"	N. 15	Removed with Paquelin. Transferred to "Hollond" Ward for hæmorrhoids.
	V	E. B.	56	Cured	N. 23	Scalpel and Paquelin.
DISORDERS OF MENSTRUATION:						
Suppresio mensium	I	E. R.	27	Relieved	M. 51	From cold.
Dysmenorrhœa	I	E. C.	31	"	M. 53	
	II	M. S.	35	"	M. 163	
Menopause	I	A. A.	42	"	N. 21	Menorrhagia.
	II	S. L.	36	"	N. 72	Amenorrhœa for the last eleven years.
DISORDERS DURING AND AFTER PREGNANCY:						
Pregnancy	I	M. W.	46	Cured	M. 77	Delivered of female child; footling. Complicated by fibroid in anterior wall of uterus.
	II	F. L.	32	"	N. 66	Delivered of eight months male child. Complicated by larny-

						geal stenosis; tracheotomy was subsequently performed owning to urgent dyspnœa.
Puerperal Eclampsia ...	I	E. W.	20	Death	M. 169	Death day after admission.
Ruptured Perineum.....	I	J. P.	27	Relieved	M. 115	Operation successful. Uterus prolapsed. Pessary inserted.
	II	F. M.	23	"	M. 200	Rupture complete. Operation successful.
Sequelæ of Abortion ...	I	J. R.	39	Cured	M. 103	
	II	L. W.	22	Relieved	M. 105	
	III	C. S.	30	"	M. 217	
	IV	J. D.	35	Cured	N. 1	Abortion result of a fall.
	V	E. W.	30	Relieved	N. 12	
	VI	J. B.	23	"	N. 41	
	VII	J. B.	30	"	N. 57	Uterus prolapsed. Second degree.
Abortion.....	I	C. C.	24	"	M. 123	Aborted day after admission, five months fœtus.
PELVIS;						
Pelvic Neuralgia	I	M. C.	48	"	M. 84	
Pelvic Peritonitis.....	I	E. B.	34	"	M. 177	Chronic.
Pelvic Cellulitis	I	B. C.	34	"	M. 93	Puerperal. Abscess formed, opened.
	II	A. W.	30	"	M. 189	Puerperal.
	III	E. H.	26	Death	M. 202	Puerperal. Large pelvic abscess. Opened per vaginam.
Pelvic Hæmatocele	I	K. F.	29	Relieved	M. 142	? Ruptured tubal pregnancy. Tumour within 2 inches of umbilicus.
	II	J. C.	30	Cured	N. 31	
	III	A. G.	29	Relieved	N. 49	Tumour 2 inches from umbilicus.
UNCLASSIFIED:						
Tubercular Peritonitis...	I	S. H.	18	"	M. 146	
	II	L. B.	21	"	M. 183	Abdominal section. Peritoneal cavity washed out.
Abscess of Groin	I	E. C.	25	Cured	M. 140	Opened and drained.
Hæmorrhoids.....	I	E. R.	38	"	M. 222	Removed.

THE GUTHRIE SOCIETY.

STATEMENT FOR 1889—90.

NINE meetings of the Society have been held during the year ending July 31st, 1890, the total number of attendances amounting to 270, averaging 30 for each meeting.

The number of members for the past year was 104, 25 new members having been enrolled.

The subscription for Life Membership has been reduced to 15*s.*, payable in one sum, or four annual payments of 5*s.* each. By this arrangement all students are enabled to become life members before they finish their course at the Westminster Hospital.

The nucleus of a Library has been founded, consisting of about forty volumes, comprising works by Darwin, Sir J. Lubbock, Herbert Spencer, Beddoe, &c.

A set of eighty typical Medical and Surgical Pathological specimens has been purchased for the use of members preparing for the final examinations.

Dr. Hall has presented to the Society a box of Histological specimens, and Mr. Yearsley has presented a portrait of Mr. Guthrie.

One of the objects of the Society is to afford students the opportunity of cultivating the art of public speaking and debate; it is therefore very gratifying to note that out of eleven papers read before the Society during the past year, ten were read by students. This is encouraging for the future, and the more so that those members who have already read a paper have expressed their desire or intention to do so again, thereby showing that they consider the time and trouble expended has not been wasted.

There have been three smoking concerts and two exhibitions of microscopical specimens.

At the Annual General Meeting, held on July 16th, Mr. C. Stonham was elected President, and Mr. A. W. Harrison Vice-President for the ensuing year.

At that meeting Dr. Sturges, in an address to the Society, pointed out that its numbers and the attendances prove the Guthrie Society to be a representative body. In reviewing the work of the Society, he said, "We are not exclusive, narrow, or bigoted. Whatever agitates mankind has agitated us. No one can complain of want of variety; we have touched many things, and, I hope, adorned them all. Chemistry and Electricity; Birds' Eggs and Monumental Brasses; Field Sports and Morality; the Ancient City of Westminster and the recent epidemic of Influenza; Phrenology and Insanity; Paracelsus and the Philosopher's Stone; these are a few of our brief studies of great subjects. You have thus had before you town and country; health and disease; science and its counterfeits; reason and folly; the rights of man and the wrongs of the lower animals. But for the rule of the Society that forbids it, no doubt we should have tackled Religion and Politics as well. And considering the times in which we are living, the place in which we assemble and its associations, there is large temptation to tackle both."

Dr. Sturges had been President of the Society for the whole of its new career, a period of nearly three years. He had been present at almost all the Committee and Monthly Meetings during the whole of that time, and no one has done more than he has to further the interests and promote the welfare of the Guthrie Society.

H. A. STONHAM, Hon. Sec.